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ON

PERIPHERAL NEURITIS.

A TREATISE.

BY

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With Illustrations.

LONDON:
CHARLES GRIFFIN AND COMPANY, LIMITED,
EXETER STREET, STRAND.
1893.

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PREFACE.

The early death of my friend and colleague, Dr. James Ross, has deprived the profession of a great part of the results of his far-reaching investigations on diseases of the nervous system, but the demand for his published works, and the acknowledged value of all his writings, indicate clearly the importance of preserving such further material as is still to be obtained.

Actuated by the desire to preserve in permanent form Dr. Ross's work upon peripheral neuritis, and urged by some of his friends to undertake the task, I have collected in this volume a series of observations which were to have formed part of a complete monograph.

Unfortunately his work was cut short by death, and, as no papers were left which in any way carried it beyond the point at which the original essays cease, it became necessary for me to write the whole of the latter part of this book.

As the volume now stands the first 263 pages are verbally Dr. Ross's own, but for the subsequent part, beginning with "Paralysis after Typhoid Fever," I am solely responsible. One exception, however, is to be mentioned. My friend Dr. R. T. Williamson has done such good work in the pathology of diabetes that I asked him to write the section relating to neuritis in that disease (pp. 361 to 375), and my best thanks are due to him for the care and thought he has bestowed on the subject. I am also indebted to Dr. Goodfellow, Resident Medical Officer to the Manchester Royal Infirmary, for much help in correcting the proof sheets; and to Mr. Coutts, House Physician to the Infirmary, for the excellent index he has prepared.

Whether the earlier pages would have been in any way modified had my colleague lived, I cannot say, but from personal knowledge of his views I believe that any alterations would have been of minor import.

In preparing this work it has been my endeavour to carry out as far as possible the original scheme, and to provide as complete a review of peripheral neuritis as our present knowledge will permit. If in the section dealing with "some points in the general pathology of peripheral neuritis," I have seemed to go beyond the rigid definitions laid down at the outset by Dr. Ross, it is because I am convinced that it is only by taking a wide view of the subject of peripheral neuritis that we can hope to give a satisfactory explanation of its complicated bearings.

JUDSON S. BURY.

10, St. John Street, Manchester.
December, 1892.

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ON PERIPHERAL NEURITIS.

Neuritis means, of course, inflammation of nerves, whilst the qualifying word *peripheral* is intended to limit our subject to inflammation of the nerves after they have passed through the intervertebral and cranial foramina. Those degenerations of the nerves, therefore, which are consecutive to disease of the motor ganglion cells and of the anterior roots are excluded from our consideration. But the subject, as thus limited, is too wide for our present purpose, inasmuch as we do not intend to consider the local diseases of individual nerves, our object being to pass in review the state of our present knowledge respecting that particular form of peripheral neuritis in which a considerable proportion of the nerve-trunks of the body are gradually invaded by disease. In order to express the fact of the implication of numerous nerves in different parts of the body the word *multiple* has been introduced, so that the full title of these diseases is *peripheral multiple neuritis*, or briefly *multiple neuritis*, or *polyneuritis multiplex*, the latter being manifestly a redundant phrase. The term *disseminated* has likewise been used to express the same fact, and the disease has thus been called *disseminated neuritis*. A further peculiarity of this disease is, that the muscles of the distal segments of the extremities are the first to lose motor power, while those of the proximal segments, and finally those of the trunk, are successively invaded. To express this mode of invasion, the word *ascending* has been used, and the disease has been named *ascending paralysis*. Another characteristic of this form of neuritis is that the disease generally attacks a few of the nerves at first and then gradually increases in extent and intensity. To give expression to this peculiarity the word *progressive* has been used, and the disease has been called *progressive multiple neuritis*, or briefly, *neuritis progressiva*. A final characteristic of this affection is that the corresponding nerves on the two sides of the body are often almost simultaneously attacked, although the disease is frequently more pronounced, at least for a time, on one side than on the other. This peculiarity is expressed by the word *symmetrical*, and the disease has consequently been named *symmetrical multiple neuritis*. These names, imperfect though they be, serve to impress upon the mind a few of the leading characteristics of the affection which is to be the subject of our remarks, and now we proceed to give a brief historical account of the growth of our knowledge of it.

History.—Peripheral neuritis has, like other diseases, a clinical and an anatomical aspect. The clinical features of the form of paralysis caused by multiple neuritis has been described from time to time by various observers, but I believe Graves* was the first who suspected that the cause of certain forms of generalised paralyses was to be found in inflammation, not of the spinal cord or higher nerve centres, but of the nervous cords themselves. "One of the most remarkable examples of disease of the nervous system commencing in the extremities, and having no connection with lesions of brain or spinal marrow," he says, "was the curious *epidémie de Paris*, which occurred in the spring of 1828. Chomel has described this epidemic in the ninth number of the *Journal Hebdomadaire*; and having witnessed it myself in the months of July and August of the same year, I can bear testimony to the ability and accuracy of his description. It began (frequently in persons of good constitution) with sensations of pricking and severe pain in the integuments of the hands and feet, accompanied by so acute a degree of sensibility that the patients could not bear these parts to be touched by the bedclothes. After some time—a few days, or even a few hours—a diminution or even abolition of sensation took place in the affected members; they became incapable of distinguishing the shape, texture, or temperature of bodies, the power of motion declined, and finally they were observed to become altogether paralytic. The injury was not confined to the hands and feet alone, but, advancing with progressive pace, extended over the whole of both extremities. Persons lay in bed powerless and helpless, and continued in this state weeks or even months."

After describing how the disease often terminated in death, how at other times patients recovered their general health but remained permanently paralysed, and how in the majority of instances, however, complete recovery took place, but only in a very slow and gradual manner, and often with frequent interruptions by relapses, the author proceeds: "The French pathologists, you may be sure, searched anxiously in the nervous centres for the cause of this strange disorder, but could find none. There was no evident lesion, functional or organic, discoverable in the brain, cerebellum, or spinal marrow. Now, here is another remarkable instance of paralysis creeping from the extremities towards the centre. Here is a paralysis affecting all parts of the extremities as completely as if it had its origin in the central parts of the nervous system; and can any one, with such palpable evidence before him, hesitate to believe that paralysis or even hemiplegia, without any lesion of the brain or spinal cord, may arise from disease commencing and originating in the nervous extremities alone?"

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* Graves (Robert J.). "Clinical Lectures on the Practice of Medicine." (1st Edition, 1843.) New Syd. Soc., 1884, Vol. I., p. 578.

In Ollivier's work[*] two cases of extensive paralysis beginning in the extremities are reported, which the author believed to have been caused by congestion of the spinal cord, but which are manifestly examples of neuritis. The first edition of Duchenne's great work appeared in 1855,[†] and examples of this form of paralysis are recorded under the name of "Paralysie générale spinale," the author believing that the symptoms were caused by a spinal affection, even although no naked eye morbid changes were discovered in the spinal cord of the one case in which alone a post-mortem examination was obtained.

In 1859, Landry[‡] published under the name of "Paralysie extenso-progressive," or "Paralysie ascendante aigue," ten cases of a rapidly invading paralysis; and in one of the cases which proved fatal on the ninth day, no lesion could be discovered either in the brain or spinal cord. The nerve trunks were not examined, but the author is inclined to believe, from the absence of bed-sores, genito-urinary disorders, and rachialgia, that they were the seat of the lesion. We have no hesitation in adopting this view, and in regarding Landry's paralysis as an acute form of multiple neuritis; but this opinion must find as much justification as we can give it when we come to subject the recorded cases to special analysis. The opinion that certain forms of widely distributed paralysis with wasting is caused by disease of the nerves—hitherto a mere conjecture—was destined, in 1864, to be placed upon the sure foundation of pathological observation. In this year, Duménil,[§] of Rouen, published the first case of the disease in which the presence of neuritis was verified by an autopsy. The importance of this case is so great as to warrant us in giving an abstract of it.

A man, aged 61 years, a tailor, after suffering from prickling of the toes for two weeks, was seized, on the 20th July, with weakness of the left arm and right leg, and some days later, of the left leg. Five days later he was scarcely able to walk or even to stand. The paralysed legs were flaccid and completely powerless, while the thighs could be moved freely. Anæsthesia was present over the whole of the right leg, but was most marked in the sole, while on the left side the loss of feeling was limited to the outer aspect of the leg and the foot. The power of swallowing and of speech were unaffected. The muscles of the hands and forearms were much wasted. The faradic contractility was diminished in some of the affected muscles, and lost in others. The patient complained of a painful feeling of numbness in the paralysed limbs, which extended over the whole of the leg and foot in the lower, but was limited to the hands in the upper extremities. The patient became progressively worse, and died four and a half months from the commencement.

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[*] Ollivier (C. P.). L'Traité des maladies de la moelle Epinière, 3me Edit., Tome II. (Obs. LXXVI. and LXVII.). Paris, 1837, p. 67.

[†] Duchenne. "L'Electrization Localisée." Paris, 1855, p. 610; 3rd Edition, 1872, p. 458.

[‡] Landry (O.). "Note sur paralysie ascendante aigue."—*Gaz. Hebdom.*, 1859, pp. 472 and 486.

[§] Duménil (Dr. L.). "Paralysie periphérique du mouvement et du sentiment, portant sur les quatre membres—Atrophie des ramaux nerveux des parties paralysées."—*Gazette Hebdomadaire*, Tome I, 1864, No. 13, p. 203.

Autopsy.—The muscles were found atrophied, pale, and small; but on microscopical examination the transverse striation was well marked, and the fibres had not undergone fatty degeneration. The small nerves of the hands and feet (median and plantar nerves) were found to have only a small number of primitive bundles, and the nerves showed an excess of connective tissue, and a large number of fat cells. In individual fibres there was a want of continuity in the myelin sheath, which, when present, manifested a marked degree of granular degeneration. The brain and spinal cord were found quite healthy.

In commenting upon this case the author remarks: "I ought not to omit to point out the resemblance in essential particulars between this form of paralysis and that which Duchenne has described under the name of *paralysie générale spinale.* In the one as in the other the disease begins in the extremities, which it attacks *en masse.* The muscles undergo a uniform wasting, and the electric contractility is abolished in muscles which have conserved their structure."

In 1866, Duméuil,* in a second communication on peripheric paralysis, reported another case of this disease with autopsy. Although this case is not such a typical example of the affection as his first, it deserves to be quoted in abstract here because it foreshadows what will be abundantly proved in the sequel, that in the diseases grouped together under the name of peripheral neuritis, the grey matter of the spinal cord is not always free from morbid changes, although the weight of the disease falls upon the nerves themselves.

M. J., aged 36 years, an ironer, was of good constitution, and free from hysteria or other nervous affection. One year before she came under observation, and after she had taken a journey of four hours in a badly constructed vehicle, in which she was squeezed so that she could only sit on the right buttock, she felt a feeling of numbness in the right gluteal region, which soon extended over the whole of the back of the limb. She suffered from continuous pains in this region, but these were interrupted by neuralgic paroxysms of great severity, during which the pains radiated to the heel, which became so sensitive that the patient was unable to place it on the ground. After a time the numbness extended over the whole of the foot, and soon afterwards the patient was unable to move the toes, and a little later the power of movement at the ankle disappeared. The muscles were flaccid and offered no resistance to passive movements. The sole of each foot, the toes, and the outer aspect of the foot and leg were completely anæsthetic, but sensation was normal on the anterior and inner surface of the leg.

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* Duménil (L.). "Contributions pour servir à l'histoire des paralysies periphériques, et spécialement de la névrite."—*Gazette Hebdomadaire de Med. et de Chir.*—Tome III., 1866, p. 52.

Soon afterwards the patient consulted Duménil (August, 1860), and she then complained of numbness of the right hand, with deep-seated pains in the whole limb. These pains were increased neither by voluntary movements, nor by pressure, and they were associated with muscular feebleness, extension of the hand being especially weak. Slight diminution of cutaneous sensibility was observed on the back of the hand, but there was no manifest muscular wasting. Eight days later the extensors of the hand and fingers were found completely paralysed. In some of the affected muscles the faradic contractility was lost, while in the others it was diminished. In November, the flexors of the right wrist and fingers lost their power, and the muscles of the fore-arm and leg of that side were decidedly atrophied. Treatment having proved of no benefit, the patient now disappeared and was lost to sight for a period of four years.

The patient came again under observation in 1864, when her condition was found not to have undergone much change, and even a slight improvement was observed in the sensibility and motor power of the right side. The patient, however, noticed that the left extremities had become implicated; she complained of lancinating pains in the plantar aspect of the great toe of the left foot, which was so sensitive that she could hardly bear the contact of the bedclothes, although there was no accompanying redness or swelling. Pressure over the course of the internal plantar nerves caused great pain. Some time later, the sole of the foot and the whole of the toes became anæsthetic, and the motor power gradually diminished. Towards the end of 1864, the patient complained of a feeling of numbness of the left half of the lower lip, and in February, 1865, of a painful feeling of numbness in the left fore-arm. A few months later, the left foot was completely paralysed, and the muscles of both hands and feet were greatly atrophied. The movements of the tongue were unaffected, but the power of swallowing was slightly interfered with, and the voice was somewhat feeble. The patient died in the month of July, from slow asphyxia, the disease having lasted about six years.

Autopsy.—The atrophied muscles showed that the primitive bundles were very small, but the transverse striation was generally well marked The external sheath of the posterior tibial, internal popliteal, and sciatic nerves was of a red colour, the nervous trunks were atrophied, and the neurilemma was somewhat thickened. On teasing a portion of nerve scarcely an intact fibre could be seen, the mass being formed of an abundant laminated tissue, in the midst of which was found a quantity of molecular granulations. In other parts pale nerve fibres were seen mixed with a large quantity of fatty globules and brilliant granulations of variable size. No part of a healthy axis-cylinder could be seen, and no defined medullary substance. A twig of the plantar nerve was seen to be grey and semi-transparent to the naked eye, and on microscopical examination it was found to consist of a laminated tissue, in the midst of which was encountered globular, fusiform, or linear fragments of medullary substance of a granular aspect. The internal popliteal and sciatic nerves were similarly affected; the nerve fibres had almost completely disappeared, and in their stead was found numerous fatty granulations.

In the spinal cord the membranes covering the posterior surface were much thickened, the posterior roots were normal, but of the anterior roots some were normal, whilst others were atrophied, and these on microscopical examination were found to be almost destitute of nerve fibres.

An examination of the spinal cord itself showed that the white substance generally was healthy, but irregular masses of fatty granules were seen to be scattered through the grey substance. The cells of the anterior grey horns were pale, and without distinct nucleus, whilst the processes were few in number, some of them being so decomposed as to be scarcely recognisable.

It is not necessary to follow Duménil's acute critical commentary of this case; it will suffice to give his general conclusion in his own words. "The existence of a peripheric paralysis, with the possibility of a progressive extension to the nerve centres, such as had been conceived by Graves, we now find to be anatomically demonstrated, and having regard to the pathological process which determines this form of paralysis, it may be named *chronic ascending neuritis.*" The view of the nature of the disease implied in this name can probably not be maintained in the light of our present knowledge; it is more probable that the main cause of the disease was the circulation in the blood of some kind of poison, like alcohol, and that the local injury to the sciatic nerve can only be regarded as a co-operating factor, or as an exciting cause. But be this as it may we are none the less sensible of the fact that Duménil was the first who laid the foundation of the doctrine of peripheric neuritis upon the basis of anatomical observation.

In his second paper Duménil* reported the clinical histories of two other cases of peripheral neuritis, in both of which the symptoms were suspiciously similar to those of alcoholic paralysis, although in the first of these cases the patient was said to have been a man of sober habits. These cases need not detain us, and it is still less necessary to analyse the six other cases he collected from literature, because the records are so imperfect that it is impossible to identify the nature of the disease with any degree of certainty, the diagnosis having been verified in none of them by a post-mortem examination.

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* Duménil (L.). *Loc. cit.* p. 85.

Cases of peripheral neuritis continued to be reported in the following years, but the pathological observations of Duménil were often lost sight of, and the clinical phenomena were attributed to disease of the spinal cord or of its membranes. A case is reported, for example, by Jaccoud,[*] and another by Vulpian,[†] the clinical descriptions of which accord well with the phenomena of multiple neuritis, but the authors regard them as instances of spinal meningitis, or meningo-myelitis. It is only justice to Jaccoud to add that, although he regarded the symptoms in the case he had himself reported as being caused by a spinal meningitis affecting the anterior roots, yet he is quite sensible of the existence of other cases of paralysis, in which the disease begins in the peripheral distribution of the nerves, as had been proved by Duménil's observations.

A case, however, is reported by Lancereaux,[‡] which the recorded symptoms and the results of a careful post-mortem examination show to have belonged to the category of neuritis. The patient, a basket maker, was the subject of acute phthisis, and, judging from the symptoms, it is not unlikely but that he may have been addicted to alcoholic excesses, but the record is silent upon this point. The patient died, and Pierret, who conducted the microscopical examination, found the spinal cord quite free from disease, but the nerves which supplied the altered muscles were in a state of advanced granular degeneration. The morbid changes in the nerves were most marked near their terminal distribution, and became less and less obvious the more the trunks approached the spinal centres.

A case is said to have been reported by Desnos,[§] about 1877, in which Pierret found that the nerve trunks had undergone extensive morbid changes, but as we have been unable to meet with the original record of the case in any of the journals of the period, it will be passed over at present.

The next important case of this disease in which an autopsy was obtained was reported by Eichhorst,[||] of Berlin, the case being described under the name of *neuritis acuta progressiva*. A female, aged 66 years, entered the clinic of Frerichs in July, 1875. Three weeks before her admission the patient was seized, without known cause, with loss of appetite and general malaise, while her bowels were alternately constipated and relaxed. Some days later she suffered from a chill, followed by a feeling of great heat, and the fever assumed a quotidian intermittent type. From the tenth to the fourteenth day the right, and two days later the left lower extremity became œdematous, and the patient complained of pain in the abdomen. The urine was scanty and of a brownish red colour.

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[*] Jaccoud (S.). "Leçons de clinique médicale." Paris, 1867, p. 372 *et seq.*

[†] Vulpian. "Malades du système nerveux." (Obs. by Labadie-Lagrave. *Union Médicale*, December 21, 1889.) Paris, 1879, p. 193.

[‡] Lancereaux et Lackerbauer. "Atlas d'anatomie pathologique." (Obs. 286.) Paris, 1871, p. 479.

[§] Desnos. Quoted by Gros (Dr. le). Contribution à l'histoire des névritis. Paris, 1879, p. 60.

[||] Eichhorst (Hermann). "Neuritis acuta progressiva."—*Virchow's Archiv*, Bd. LXIX., Berlin, 1877, p. 268.

On the second day after admission, the patient complained of severe boring pains, accompanied by sensations of coldness and formication in the right leg. The pain was continuous, but was subject to paroxysmal exacerbations of great intensity, during which the pain radiated from the knee to the tips of the toes. In the evening of the same day, the muscles innervated by the left superficial tibial nerve were completely paralysed, while the sensibility of the skin supplied by it was considerably lowered. The leg felt hot, it was somewhat red, and was covered by a profuse sweat, while the faradic contractility of the nerve and affected muscles was lowered. On the following day the leg was of normal temperature and colour, but the skin and muscles supplied by the affected nerve were completely anæsthetic and paralysed, and both the nerve and muscles failed to respond to the faradic current. At the end of six days, the pain, with its attendant phenomena of coldness, formication, and sweat, recurred during the night, and on the following day the muscles supplied by the left anterior tibial nerve were found in the morning to be slightly, and in the evening completely paralysed, while the faradic contractility, diminished at first, was soon lost. The same night another paroxysm of pain ushered in paralysis of the muscles supplied by the posterior tibial nerve, so that now all the movements of the foot were completely lost. The disease made rapid progress. The patient suffered from intense lancinating and boring pains in the other extremities, and each paroxysm of pain was promptly followed by paralysis of the muscles supplied by the motor branches of the affected nerves, so that within ten days from the commencement of the nerve affection, all the limbs were completely paralysed and anæsthetic. The course of the disease was marked by a moderate degree of fever, and albumen, absent at first, appeared in the urine. On the evening of August 15th—three weeks after admission—the patient became suddenly blind, and the pupils were dilated and reactionless to light, but the optic discs were found normal on ophthalmoscopic examination. The other special senses were unaffected. There were no sensory disorders of the trigeminus, and no paralysis of the facial muscles. Slight difficulty of articulation was noted, but the tongue did not deviate to either side on protrusion, and there was no difficulty in swallowing. The extremities were remarkably warm, œdematous, and covered with profuse sweat.

There was involuntary discharge of urine, and the bladder was emptied by means of the catheter. During the night, profound coma supervened, the breathing was irregular but not laboured, the pulse continued regular, and the patient died on the following morning.

Autopsy.—The usual signs indicating a moderate degree of chronic interstitial nephritis were found, and the heart was somewhat hypertrophied. No focal disease was discovered in the brain, and the spinal cord and its membranes were normal. The optic chiasma was of an intensely red colour, and its under surface was covered by fine, tortuous, and congested vessels; both the chiasma and optic nerves were found on section to be of a bright rosy tint, and studded with red points. The nerve trunks of the arm and forearm and the left tibial nerve, when examined with the naked eye, were seen to be of an intensely red colour; on microscopic examination the vessels of the perineurium were found enormously distended and tortuous, their walls were thickened, their nuclei were increased in number, and they were surrounded by lymphoid cells, which infiltrated the neighbouring connective tissue. Extensive changes were met with in the endoneurium and extravasations of blood, which compressed the nerve fibres, were not unfrequently observed. The nerve fibres and especially those lying next the endoneurium, showed marked degeneration. The medullary sheath was broken into masses of variable size, and the axis-cylinders and nuclei presented the usual appearances observed in nerve degenerations.

In 1879, Joffroy* reported the case of a washerwoman, aged 33 years, who was admitted into hospital on March 5th, whilst suffering from advanced phthisis. It was only on the previous month that she first noticed that her legs were becoming feeble, and on her admission the patient was unable to walk or raise her feet from the bed. Two weeks after her admission the arms also became feeble and were completely paralysed at the end of ten days. The faradic contractility was diminished in all the affected muscles. There was great mental feebleness; but throughout the whole course of the disease sensory disorders were not prominent, except that the muscular sense was lost. There were no disturbances of the urinary functions, and no bed-sores. The patient died on April 7th.

Autopsy.—The spinal cord and its membranes, as well as the anterior roots, were found healthy on microscopic examination, but the sciatic, radial, and ulnar nerves showed marked signs of parenchymatous inflammation. The muscles were atrophied, and in a state of fatty degeneration. The membranes of the brain manifested the appearance of a chronic inflammation, which explains the presence of mental symptoms. In this communication Joffroy distinguishes three forms of neuritis, viz., (1) Spontaneous parenchymatous neuritis (*neuritis a frigore*, rheumatic neuritis); the multiple variety of this form he named generalised parenchymatous neuritis; (2) saturnine neuritis; and (3) neuritis following infectious diseases, such as typhus, variola, and diphtheria.

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* Joffroy (A.). "Archiv. de Physiologie," 1879, p. 172.

Soon after the appearance of Joffroy's case a very important service to the doctrine of neuritis was performed by Gros,* who made it the subject of an excellent thesis in which all the cases of multiple neuritis hitherto reported were collected and analysed.

The theory of a multiple neuritis to account for certain forms of generalised paralysis was now assuming a definite form, and the next great advance towards its completion came from Germany. In 1880 a carefully recorded case of neuritis, with autopsy, was published by Leyden,† and a second case‡ in the same year. But the service which Leyden rendered to the doctrine of neuritis is to be found, not so much in the publication of these cases, as in the searching criticism to which he subjected the views which had led the majority of neurologists to seek for the cause of every form of generalised muscular atrophy in disease of the anterior grey horns of the spinal cord (poliomyelitis anterior). The cases themselves are, however, so important as to deserve to be mentioned here in brief abstract.

A sailor, aged 28 years, entered the hospital on February 7th, 1878. Three days before admission he was, after exposure to cold, suddenly seized with chilliness and fever, which were accompanied by severe pains and great weakness of the extremities. On admission the urine contained a large quantity of albumen. The legs and forearms, especially in the neighbourhood of the knees and elbows, showed a white œdematous swelling, and pressure over the muscular masses of the extremities and abdomen caused great pain, which was also elicited by all efforts at spontaneous movements. The patient also suffered greatly from tearing pains in the extremities, even in the absence of pressure or movements, and these were accompanied by a distressing feeling of formication. The cutaneous sensibility was found much diminished on objective examination. The feebleness of the extremities first noticed soon advanced to decided paralysis with atrophy of the muscles attacked, the extensors of the forearms and legs being affected in a predominating degree. The patellar-tendon reactions were lost on both sides, and the nerves and muscles gave the reaction of degeneration to electrical examination. The nails were of a yellow colour, broken, and strongly curved. In the course of a few months a gradual improvement took place in the motor power of the lower extremities, but in the upper extremities the forearms and hands remained permanently paralysed, and the hands assumed the form of the "claw hand." The kidney disease first noticed, however, gradually progressed, and the patient died eleven months from the commencement of the symptoms. The diagnosis was rheumatic fever with *multiple* (*symmetrical*) *neuritis*.

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* Gros (Dr. le). "Contribution à l'histoire des névrites." Paris, 1879.

† Leyden (E.). "Ueber einen Fall von multipler Neuritis."—*Charité Annalen*, Berlin, 1880, p. 206.

‡ Leyden (E.). "Ueber Poliomyelitis und Neuritis."—*Zeitschrift für klinische Medicin*, Berlin, 1880, p. 307.

Autopsy.—The spinal cord was normal, and a microscopic examination failed to discover any morbid changes in the ganlion cells of the anterior horns, or in the anterior roots. The muscles were greatly atrophied. The radial nerve was found swollen at the level of the elbow on each side, and on microscopical examination it was seen that this thickening was caused by an accumulation of fat cells around the nerve bundles, which themselves were much degenerated. The nerves of the lower extremities manifested no marked changes.

The second case was that of a merchant, aged 31 years, who had suffered for two or three years before he entered the hospital, from occasional attacks of loss of consciousness, in some of which he fell to the ground. About Christmas, 1878, he began to suffer, quite independently of these attacks, from formication of the toes and anterior part of the sole, first of the left, and soon afterwards of the right foot. On July 6th, 1879, the patient had an epileptiform seizure, which passed off as usual without leaving a trace behind; but four days afterwards, on getting up he felt a remarkable heaviness of the lower extremities, which increased so rapidly that he took to his bed and was unable to get up afterwards. About this time he complained likewise of formication in the fingers and hands, and his sense of touch was much diminished.

On July 17th, 1879, the patient entered the hospital, and it was then found that sensation was nearly abolished in the peripheral parts of the extremities, and he complained of a burning pain in the left hand and inner side of the left knee. The lower extremities were almost completely paralysed, and the extensors of the hands and fingers were very feeble; the muscles were flaccid and tender to pressure, and the tendon reactions were lost, while the nerves and muscles gave the reaction of degeneration on electrical examination. The temperature was normal, and the pulse 70, but there was slight jaundice, and the liver was tender. The paralysis progressively increased in intensity and extent, the muscles rapidly wasted, and the sensory disturbances continued unabated and were now associated with a feeling of great coldness of the hands and toes. On August 28th the temperature rose, the pulse was 124 in the minute, the patient suffered from headache and great difficulty of breathing, and the body was covered by a profuse sweat. These symptoms continued until September 3rd, when the patient died. At the end the breathing was purely costal, the abdomen being drawn in during each inspiration.

Autopsy.—The spinal cord was found normal, with the exception of slight and unimportant changes in the ganglion cells of the anterior horns. The nerve trunk manifested in a high degree the signs of degenerative atrophy.

In 1881, Dr. Grainger Stewart* reported three cases of the disease which have acquired a noteworthy importance because of being the first recorded in this country in which the diagnosis of peripheral neuritis had been made.

Two of these cases, after manifesting the usual sensory phenomena in conjunction with atrophic paralysis of the distal segments of the extremities, improved slowly under treatment, and the patients recovered. The third case deserves to be quoted in brief abstract, owing to the special significance it has acquired, chiefly because of the care with which the nerve tissues were examined by Professor Hamilton, now of Aberdeen University, but then holding the post of pathologist to the Royal Infirmary, Edinburgh. G. F., aged 31 years, a hotel keeper, entered the Royal Infirmary, November 8th, 1880. In the beginning of the previous August, he noticed a weakness in his legs, and a dancing of objects before his eyes. A few weeks later pain of a prickling character, and gradually increasing in intensity, came on in the legs, and subsequently in the hands, these pains being accompanied by loss of power and stiffness. On admission he complained of tingling pains in both legs; these were accompanied by numbness, and a feeling of coldness in the feet and hands; sensibility to heat, tickling, and pain was diminished; while the conductivity of impressions was delayed, and he had a difficulty in localising them. Muscular sense was diminished in the left foot, and absent in the right foot, but was normal in the hands. The cutaneous reflexes were absent in the soles, but normal in the cremaster and abdomen. The patellar-tendon reflex was absent in both legs. Voluntary motion was greatly impaired in legs and hands, and the fingers remained in a semi-flexed position. Electric sensibility and irritability were much diminished in the legs and forearms, and attempts to use the muscles produced pain. There were no vaso-motor or trophic changes. The patient was drowsy, and his memory imperfect. He improved a little under treatment at first, but a month after admission he died of double pneumonia.

Autopsy.—The most noteworthy gross lesion observed was red hepatisation of the lower lobes of both lungs. On microscopical examination the median, ulnar, and tibial nerves were found in a state of advanced fatty degeneration, but the details of the appearances observed need not be described at present. Slight evidences of secondary degeneration were observed in the columns of Goll, and in the direct cerebellar tracts, but the grey matter and anterior roots were normal.

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* Stewart (T. Grainger). "On paralysis of hands and feet from disease of nerves."—*Edinburgh Medical Journal*, Vol. XXVI, Edinburgh, 1881.

It is unnecessary to pursue at present the history of the development of the doctrine of multiple neuritis any further in this direction, but we will go back to trace briefly two or three other lines of investigation which helped to place it upon a comprehensive as well as a sure foundation.

As far back as 1862 Charcot and Vulpian found,[*] in a case of diphtheritic paralysis, that the nerves of the palate had undergone a degeneration characterised by the medullary sheath being broken up into globular masses and granules, just as occurs in the peripheral end of a nerve after section. These observations were soon after confirmed by Lorain and Lépine,[†] and similar changes were observed by Liouville, in the phrenic nerve. Other observations tending to prove that in diphtheritic paralysis the primary lesion is to be found in the peripheral nerves, soon followed, but these need not detain us.

A similar history applies to the morbid anatomy of lead paralysis. In 1871 Lancereaux[‡] described changes which he had found in the affected muscles and in the nerves which supplied them in a fatal case of lead paralysis. In the following year these observations were confirmed and extended by Gombault and Charcot,[§] and soon afterwards by Westphal,[¶] and although Vulpian and others reported the occurrence of morbid alterations of the ganglion cells of the anterior horns, the opinion was gaining ground amongst pathologists that the nerve trunks themselves were the seat of the primary lesion in this disease.

Turning now to alcoholic paralysis, we find that, as early as 1852, Magnus Huss[‖] had been struck by the similarity existing between the clinical phenomena of the disease and the symptoms caused by chronic poisoning by lead, arsenic, and mercury, so that even at this early date there were not wanting considerations which tended to prove that all these diseases owned one common pathology. These observations were, however, lost sight of for a time, and it was not until twelve years afterwards that the subject received an adequate and scientific treatment at the hands of Lancereaux,[**] who, besides giving an exact description of the clinical phenomena, reported a case with autopsy in which the nerves of the extremities were found in a condition of granular degeneration.

In that case, however, Lancereaux stated that the cord itself was in a state of sclerosis, and it was not until 1881[*] that he expressed a definite opinion that alcoholic paralysis is the result of disease of the peripheral nerves. In Germany this opinion was endorsed for the first time in 1883 by Moeli,[†] who found post-mortem evidences of neuritis of the two anterior crural nerves in an alcoholic patient, while the nerve centres were free from disease. In this country the clinical phenomena of alcoholic paralysis were recognised and carefully described by several observers whose names need not be mentioned at this stage of our enquiry, inasmuch as they were led to seek for the cause of the disease either in a softening of the spinal cord, or in an inflammation of the spinal membranes. My colleague, Dr. Dreschfeld,[‡] has the merit of being the first in this country, not only to have adduced anatomical evidence in favour of the neuritic theory of alcoholic paralysis, but also to have recognised the full significance of this view in bringing together under one common pathology the paralysis caused by diphtheria, lead, alcohol, and various other poisons, as well as the cases which had been described by Joffroy, Eichhorst, Leyden, Grainger Stewart, and others, under the name of peripheral or multiple neuritis. Since the publication of Dr. Dreschfeld's contribution to the study of alcoholic paralysis, numerous papers and monographs on one or other, or on all of the groups of diseases now comprised under the name of peripheral or multiple neuritis, have appeared on the Continent, in America, and in this country, not the least valuable of these being Dr. Buzzard's[§] very able work.

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[*] Charcot et Vulpian.—*Comptes Rendus de la Soc. de Biologie*, 1862.

[†] Lorain et Lépine. Art. "Diphtherie," *Nouveau dict. de Méd. et de Chir.*, Tome II., 1869, p. 619.

[‡] Lancereaux. "Note relative d'un cas de paralysie saturnine avec altérations des cordons nerveux et des muscles paralysés."—*Gaz. méd. de Paris*, 1862, p. 707 ; and 1871, p. 383.

[§] Gombault. "Contribution à l'histoire anatomique de l'atrophie musculaire saturnine."—*Arch. de Physiologie*, Tome V., 1873, p. 592.

[¶] Westphal. "Ueber eine Veräderung des N. radialis bei Bleilähmung."—*Arch. f. Psychiatrie*, Bd. IV., Berlin, 1874, p. 601.

[‖] Huss (Dr. Magnus). "Chronische alkoholskrankeit oder Alcoholismers chronicus."—Stockholm and Leipzig, 1852.

[**] Lancereaux. Art. "Alcoölisme."—*Dict. Encyclop. des Sc. Med.*—Tome I., Paris, 1864

One of the most important results obtained from all these observations is the knowledge that multiple neuritis is almost invariably caused by the action of some kind of poison. In one case it owns for its cause alcoholic excess; in another poisoning by lead, arsenic, or some other metal; in a third it is a sequel of diphtheria, the exanthemata, typhoid fever, or syphilis; in a fourth it has resulted from gout or rheumatism, and so on. So uniformly is the disease being now traced either to the action of some kind of poison or to some profound cachexia that it is very doubtful whether or not such a division as spontaneous or idiopathic neuritis exists.

At the same time a few cases are still met with in which it is difficult or impossible to trace the action of a definite poison, so that in our classification of the disease we shall still retain the division of idiopathic peripheral neuritis, giving under this name a general description of the symptoms which are more or less common to all the varieties of the disease. Regarding, as we do, Landry's paralysis as an acute form of peripheral neuritis, we shall treat of it along with the so-called idiopathic variety. At the same time it must be acknowledged that hardly a single case of this disease has ever been recorded, the origin of which cannot be traced to the action of some kind of poison; now it is due to alcohol, and again it is a sequel to some fever, to syphilis, or to inoculation with the poison of rabies. Landry's paralysis, therefore, instead of being regarded as a division of peripheral neuritis in its own right, ought to be distributed amongst the other divisions. But this disease has for so long held such an important and honourable position in our nosology that a separate treatment of it in a division of its own, or rather in a sub-division of idiopathic peripheral neuritis, is a necessary preliminary to its being distributed among the other divisions of neuritis. Another circumstance which gives rise to what logicians call a cross division is the fact that the symptoms are sometimes caused by the combined action of two or more poisons. Lead paralysis is, for example, frequently met with in drunken painters, and it is rare to meet with gouty neuritis without a possible complication with alcohol, so that it is difficult to estimate how much of the phenomena is to be attributed to the action of one poison, and how much to the action of the other. A still further complication is encountered when we endeavour to estimate how much of the disease is to be attributed to the action of a particular poison, and how much to an inherited weakness of the nervous system itself, for it is well known that some persons are much more liable to be injuriously affected by certain poisons than others. One other difficulty remains to be mentioned. In certain cases of peripheral neuritis it is quite certain that the disease has resulted from the action of a very definite poison, but the nature of the poison has not been ascertained beyond dispute. In cases of paralysis arising during the inhalation of the fumes of bisulphide of carbon, it is doubtful whether the symptoms are caused by the action of this gas itself, or by sulphuretted hydrogen formed by its decomposition. And in the paralysis which is caused by the inhalation of the fumes of the new explosive agent named roburite, it is a matter of doubt whether the symptoms are caused by the di-nitro-benzine, of which it chiefly consists, or by such products as carbon monoxide, formed during its combustion. These remarks show that it is not possible for us to sketch out a classification of the various forms of peripheral neuritis, which will not be marred by serious defects. In the first place, it will have the serious defect of containing cross divisions; in the second place, the facts of neuritis as we meet them in nature, are too complicated and variable to find an adequate expression in any one classification; and, in the last place, the etiological divisions at present adopted will have to be replaced and supplemented, as our knowledge of the disease advances; and as this knowledge is rapidly increasing, so must our classification be subject to equally rapid alterations.

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[*] Lancereaux. "Etudé sur altérations produit par l'abus des bois alcoöliques."—*Gaz. Hebd. Méd.*, Paris, 1868, pp. 438 and 464.

[†] Moeli (A.). "Alcoholismus, &c. Atropische Lähmung der Extensoren am Oberschankel."—*Charité Annalen*, 1883, VIII. J. pp. 552; and "Statistiches und klinischer über Alcoholismus."—*Charité Annalen*, IX., Jahrg, p. 541.

[‡] Dreschfeld (J.) "On alcoholic paralysis."—*Brain*, 1884, p. 200.

[§] Buzzard (T.). "On some forms of paralysis from peripheral neuritis." London, 1886.

Subject to the above reservations, then, we submit the following classification of the different forms of peripheral multiple neuritis:—

I.—The idiopathic form—
  (1) Acute (Landry's paralysis).
  (2) Sub-acute.
  (3) Chronic.
II.—The toxic form—
  (1) *Diffusible stimulants:* Alcohol, carbon monoxide, bisulphide of carbon, di-nitro-benzine (roburite), aniline.
  (2) *Animal poisons:* Diphtheria, typhoid and other fevers, septicæmia, syphilis, pneumonia, tubercle, malaria, beri-beri, leprosy.
  (3) *Metallic poisons:* Lead, arsenic, mercury, phosphorus, and silver.
  (4) *Endogenous poisons:* Rheumatism, gout, chorea, the puerperal state, diabetes.
III.—The dyscrasic form: Chlorosis, marasmus, cancerous and other forms of cachexia, vascular degeneration.
IV.—Sensory, vaso-motor, and trophic neuritis—
  (1) The neuritis found in cases of ataxia, and which has been named *neuro-tabes periphérica.*
  (2) The vaso-motor neurosis, first described by Weir Mitchell under the name of *erythromelalgia.*
  (3) Raynaud's disease.
V.—The irritative form of neuritis in which spasm predominates over paralysis—
  (1) Tetany.
  (2) Professional hyperkineses.

The right of admission to the category of peripheral neuritis, even provisionally, of some of these diseases, notably those comprised in the fifth group, must be justified in the sequel.

I.—Idiopathic Peripheral Neuritis. (1) Acute Ascending Paralysis. Landry's Paralysis.

Definition.—Acute ascending paralysis is characterised by a motor paralysis, which usually begins in the lower extremities, spreads rapidly to the muscles of the upper extremities and trunk, often invades the muscles of the face, tongue, palate, larynx, or eye-balls, and either proves fatal in a short time by respiratory paralysis, or improvement begins and progresses gradually to partial or complete recovery. The general sensibility is almost always affected, but not often in a profound degree; the bladder and rectum are only occasionally implicated; and the muscles are said not to undergo atrophy, and to retain their electrical excitability.

History.—The following tables, in which many, if not all, of the recorded cases of Landry's paralysis are tabulated and arranged in chronological order, afford, even in the absence of any commentary, a general history of the development of our knowledge of this important disease. It is not pretended that the tables contain all the recorded cases, but the list is a tolerably full one—sufficiently so, we trust, to enable some trustworthy conclusions to be deduced with regard to the course and nature of the disease. A few abbreviations are used in the tables which require a word of explanation. In the columns recording the duration of the disease, I stands for the period of invasion, P for the period of advancing paralysis, and C for the period of improvement and regression of the paralysis. The dividing line between these periods must necessarily be drawn in a somewhat arbitrary manner. Vague descriptive terms like "lassitude" and "heaviness of the limbs" are held to belong to the period of invasion, and when mention is made in the record of the presence of a special weakness of any part of the body, it is held that the stage of paralysis has begun. Such terms are, however, often merely the patient's description of the subjective feelings which accompany the minor degrees of paralysis, so that, as we have just said, the division between the stage of invasion and that of paralysis is, in many of the cases at least, a purely arbitrary one. The stage of convalescence is held to have begun at the first mention in the record of improvement having been observed, or at the first indication that the paralysis has ceased to progress, so that the dividing line between the stages of paralysis and convalescence is generally fairly well marked. It is, however, much more difficult to fix the end of the period of convalescence than its beginning, and it is frequently quite impossible to do so. In Case 32 for example, the stage of convalescence is stated to have lasted eight weeks, but this only indicates the end of the observers's knowledge of the progress of the case, for on looking at the column which gives the various terminations, it will be seen that the lower extremities of the patient were still feeble when he disappeared from the author's notice. Another difficulty we have to contend with is that the stage of convalescence is sometimes interrupted by an intercurrent disease which may prove fatal. In Case 54 for example, the stage of paralysis is said to have lasted over the unusually long period of a month, while the stage of convalescence is only eighteen days. But on turning to the termination of the case, it is seen that the patient died from an acute attack of gastro-enteritis. It is hardly necessary to state that in the column in which the electrical re-actions are given, C stands for contractility, and R.D. for the reaction of degeneration, or to point out that the number which the case occupies in the tables corresponds to the number referring to the name of the observer in the Bibliography given at the end.

Observer.	Age.	Sex.	Duration.	Causation.	Sensory Disorders
1.—Ollivier	31	F.	I.—24 hours. P.—3 days.	Began month after normal confinement.	Formication of fingers and feelings of coldness of extremities. Lancinating pains in legs.
2.—Ollivier	41	M.	P.—5 days. C.—9 months.	Journeyed three or four hours in a boat on a cold night.	No sensory disorders mentioned.
3.—Walford (T. L.)	17	M.	I.—2 days. P.—10 days.	Slept in an out-house in wet clothes.	Fidgets ....
4.—Walford (T. L.)	..	M.	P.—Few days.	Exposure to cold.	Pain along sciatic nerves. Numbness of extremities.
5.—Brochin	23	F.	P.—10 days.	Attack of confluent smallpox, beginning Dec. 25, and convalescent Jan. 6.	Jan. 7.—Taken suddenly with acute and burning pains in neck, passing down vertebral column. Anæsthesia of lower extremities.
6.—Landry (O.) ..	43	M.	I.—6 weeks. P.—8 days.	Several febrile attacks, the last of which was accompanied by pains in the side. Treatment by 3 bleedings, emetics, and blisters. Imperfect recovery.	Numbness and tingling of fingers and toes, and coldness of extremities for six weeks.
7.—Landry (O.) ..	..	..	P.—5 days. C.—2 weeks.	Convalescence, after long attack of typhoid fever.	....
8.—Landry (O.) ..	..	F.	P.— C.—7 days.	Suppression of menses and exposure to cold.	..
9.—Landry (O.) ..	..	F.	P.—3 days, with subsequent relapses.	Suppression of menses from moral shock.	....
10.—Landry (O.) ..	..	..	P.—Very acute. C.—Slow and imperfect.	Exposure to cold	....
11.—Landry (O.) ..	..	..	P.—Very acute. C.—Slow, but complete.	Exposure to cold	....
12.—Landry (O.) ..	..	F.	P.—2 days.	After confinement................	....
13.—Landry (O.) ..	..	..	P.—Slow.	Syphilitic diathesis	..
14.—Landry (O.) ..	..	F.	P.—Intermittent.	....	....
15.—Landry (O.) ..	..	..	P.—	....	....

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
Next morning found her limbs feeble, and two days later, legs almost completely paralysed 3rd.—Difficulty of deglutition, dyspnœa and death.	....	....	Death from asphyxia.	Congestion of cerebral and spinal membranes.
Felt feeble same night, next morning unable to stand. 2nd day all movements paralysed, except lateral movements of the head. Dyspnœa. Gradual improvement after 15 days.	....	....	Partial recovery in nine months.	....
In two days after exposure difficulty in moving legs. Next day arms weak, then followed difficulty of articulation, and whispering voice, dyspnœa and death.	....	....	Death from asphyxia.	Spinal cord softened in parts. Theca vertebralis contained much fluid.
Paralysis gradually creeping upwards, and death by suffocation.	....	....	Death from asphyxia.	....
January 7.—Complete paralysis of lower extremities, and of abdominal muscles. Left arm affected. Respiratory difficulties. January 15.—Entered hospital under Trousseau. Dyspnœa, and, Jan. 17, death.	....	Bladder distended. Constipation, relieved by croton oil.	Death from asphyxia.	Lasegne found no morbid changes in brain or cord or their membranes.
Three or four months after last fever patient felt general weakness, which gradually increased to paralysis, first of legs, then successively of upper extremities, trunk, and tongue. Finally paralysis of diaphragm, dyspnœa and death.	Reflex actions lost Faradic C. normal.	....	Death from asphyxia.	No lesion in brain or spinal cord. Nerves not examined.
Ascending paralysis reaching maximum intensity in five days.	....	....	Recovery in two weeks.	....
Ascending paralysis. No mention of duration.	....	....	Recovery in seven days.	....
Paralysis generalised in three days, with threatening symptoms. Amelioration, with frequent relapses and chronic course.	....	....	Death in a sudden paroxysm after chronic course.	....
Very acute and menacing ascending paralysis.	....	....	Partial recovery.	....
Very acute and menacing ascending paralysis.	. ..	....	Complete recovery after long time.	....
Ascending paralysis fatal on second day.	....	....	Death.	....
Ascending paralysis pursuing a more or less chronic course, and rapidly disappearing under antisyphilitic treatment.	....	....	Complete recovery.	....
Intermittent ascending paralysis cured by quinine.	....	....	Recovery.	....
Ascending paralysis.	....	....	Recovery.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
16.—Liégard (A.) ..	2	..	P.—Few days. C.—3 weeks.	Attack of measles, occurring in the course of an exhausting diarrhœa.	Anæsthesia of lower extre ties.
17.—Leroy d'Etolles (M.R.)	23	F.	P.—10 days.	Jan. 6.—Convalescent from attack of confluent variola.	Jan. 8.—Anæsthesia of pa lysed parts. Jan. 16.—Lancinating pains cervical region, anæsthe of trunk, lower extremiti trunk and left arm.
18.—Pidoux (M.) ..	25	M.	P.—15 days. C.—2 or 3 months.	Attack of discrete variola, without notable sore throat. Paralysis began during dessication of pustules.	Acute pains and cramps in quadriceps of left leg, an few days later in corresp ding muscles of right leg.
19.—Macario (M.)..	49	F.	P.—Few weeks C.—2 or 3 months.	Patient ill-lodged and ill-nourished. Attack of pneumonia of right base treated by high doses of emetics and large blister. Convalescence, but blistered surface did not heal, and continued to suppurate and was covered by a white exudation.	Formication of soles of f and palms of hands. Ne day there is a progress extension of wound cau by blister, and the formi tion becomes more mark and persisted a month af the paralysis disappeared.
20.—Bauchet	..	F.	P.—4 days.	Undergoing treatment at Enghien by sulphur baths. Gastritis.	Anæsthesia extended upwa to below the arms.
21.—Kussmaul (A.)	22½	..	P.—4 days.	Exposure to cold	Numbness of finger tips toes.
22.—Kussmaul (A.)	51	M.	P.—19 days.	Syphilis of parietal bone..........	Feeling of "pins and needl in extremities. Tactile sc bility blunted. Smell minished.
23.—Gubler (A.) ..	35	M.	P.—16 days.	Double pneumonia treated by four bleedings, large doses of emetics and two blisters. The blistered surfaces continue to suppurate for a long time.	Pains in legs and arms.
24.—Gomes do Valle (A.)	25	M.	P.—13 days.	Sergeant of a regiment of riflemen. Constitution enfeebled. No syphilis. Onanism.	Formication in paralysed pa
25.—Gomes do Valle (A.)	40	M.	P.—10-12 days C.—4 months.	Captain of infantry. Acute hepatitis and yellow fever in 1857. Dec., 1859.—Double pneumonia, very protracted. March, 1860.—Dulness of base of right lung, and diarrhœa for many months. Anorexia. No fever. Convalescence.	Formication and cramps lower extremities. An thesia of lower limbs trunk. Sensation of c striction of throat and o phagus.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
At beginning of convalescence feebleness of lower extremities, next day complete paralysis of lower and great weakness of upper extremities and muscles of neck. Mastication and deglutition difficult.	....	Apathy.	Complete recovery in 3 weeks.	....
Jan. 8.—Sudden and complete paralysis of lower extremities, abdominal muscles, and left arm. Jan. 16.—Orthopnœa. Jan. 18.—Respiratory paralysis and death.	....	Retention of urine. Incontinence of fæces.	Death from asphyxia.	No change observed in the nerve centres or their membranes beyond a doubtful congestion of the vessels of the brain.
Began by nasal speech and return of fluid through nose. Veil of palate loose and flaccid. Few days later almost complete paraplegia, and at end of 15 days upper extremities feeble. Improvement, recovery taking place in reverse order.	....	....	Complete recovery in 2 or 3 months.	....
Lower extremities feeble, and next day they became paralysed, and the arms were implicated. 2½ months after disappearance of pneumonia there is complete paraplegia, and the arms are feeble, but 15 days later the patient is able to walk.	....	....	Complete recovery in 2 or 3 months.	....
Whilst at dinner seized with trembling, and sudden inability of moving her legs, and at night complete paraplegia. 4th Day.—Upward extension of paralysis, and death.	Excess of reflex movements in legs.	2nd day. — Paralysis of bladder and rectum.	Death.	....
1st day.—Great weakness of legs. 3rd.—Complete paraplegia. 4th.—Death from respiratory paralysis.	....	....	Death from asphyxia.	....
Weakness of all the limbs. External strabismus. Weakness of muscles of lips. Uvula drawn to right, profound paralysis of extremities.	Electrical reactions normal.	Dulness of both apices of lungs. Sudden discharge of stinking pus.	Death in delirium.	Syphilitic cicatrices of liver. Cerebral dura mater thickened. Cord normal.
Great feebleness of legs and arms, progressing rapidly to complete paralysis of lower and great feebleness of upper extremities.	....	Obstinate constipation.	Death.	No autopsy.
May (beginning).—Complained of weakness of legs for few days, and now unable to stand. Paralysis invaded in succession the thighs, hands, arms, trunk, pharynx, œsophagus, respiratory muscles, and, May 18th, death.	....	....	Death from asphyxia.	....
April.—Noticed that he could not raise himself from bed, or walk, and his hands so feeble that he could not undress himself. Paralysis increased; dyspnœa. Ten days later, improvement, which continued to recovery.	....	....	Recovery in 4 months.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
26.—Leudet (D.) ..	32	M.	P.—7 days. C.—Many months.	Double pneumonia treated by large doses of tartar emetic and blisters. Feb. 1st.—Convalescence. The blistered surface over the sternum ulcerated, but was not covered by white membrane. Treated early in February. March 5.—Sore throat. March 9.—Re-entered hospital. No diphtheric membrane.	March 9.—Formication of f and, later, of all the extre ties. Analgesia of outer a anterior surfaces of legs.
27.—Pellegrino-Levi (Dr.)	22	M.	I.—3 or 4 months. P.—12 days.	Free beer drinker at one time, but sober in recent years.	For a few months feeling fatigue. Numbness a tingling of fingers and to Sensibility to pain and tou preserved. All movemen caused great pain. Gr restlessness.
28.—Bablon (E.) ..	39	M.	P.—24 days.	No venereal or alcoholic (absinthe) excess, and no syphilis. July 12, coitus in erect posture, and symptoms began two days after.	Paræsthesia and numbness soles of feet and palms hands. Incomplete an thesia of lower, and, to so extent, of upper extremiti August 7.—Analgesia of pe pheral segment of extre ties.
29.—Pellegrino-Levi (Dr.	..	M.	P.—6 days.	Case of Baron Cuvier. No cause assigned.	Complained only of gene malaise and pain in e gastrium.
30.—Leudet (E.) ..	51	M.	I.—17 days. P.—13 days.	Whilst drunk attempted to commit suicide by inhaling vapour of burning charcoal.	Dec. 23.—Heavy pain, ra ating towards right butto and aggravated by pressu over sciatic nerve.
31.—Leudet	Young.	F.	P.—6 days.	Abdominal typhus and convalescence on eighteenth day, and seven days later symptoms began.	Numbness of inferior e tremities.
32.—Jones (C. Handfield).	39	M.	P.—2 weeks. C.—8 weeks.	Exposure to chills when perspiring. Sore on penis, but no constitutional syphilis.	Numb fingers and toes, sen tion of "pins and needle up to elbows, and anæsthe of lower extremities. F cold.
33.—Caussin (E. T.)	60	F.	P.—5 days.	....	Formication and pains in low extremities. Acute pain lumbar region. *Pressure region of vertebral colu painful.* Slight anæsthe of feet.
34.—Gru (M.)	8	—	P.—Few days.	Weak child. April 8.—Rigors foreboded severe disease, but nothing observed until twelve days later.	Sensibility intact.
35.—Russell (Dr.)..	60	M.	P.—Few days.	Gentleman of gouty habits. Worry and mental and bodily exertion.	Severe pain and dragging the loins.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions	Termination.	Morbid Changes.
Mar. 9.—Slight feebleness of lower extremities, treated by strychnia from April 3 to 10. No spasm, but progressive enfeeblement of legs and also of hands. April 12.—Lower extremities completely paralysed, and upper very feeble. Slight dyspnœa. April 15.—Increase of paralysis. Improvement began in movements of hands, and progressed to complete recovery in many months.	....	No fever. Constipation.	Complete recovery in many months.	....
Feeling of fatigue passed into decided weakness, and in 5 days to paralysis, complete of lower, and partial of upper extremities. This followed by diplopia, dysphagia, paralysis of diaphragm, dyspnœa, and death.	Reflex actions lost. Faradic contractility normal.	Absolute constipation. Pulse, 90—94. Profuse sweat.	Death from asphyxia.	Cornil found the cord, nerve roots, spinal ganglia, and pneumogastric nerves normal.
July 14.—Heaviness of limbs progressing to paralysis. July 18.—Station and walking impossible, and hands feeble. Difficulty in sitting up in bed. Paralysis gradually increased, and Aug. 9 was complete in lower and almost complete in upper extremities. Embarrassment of speech, dyspnœa. Death from asphyxia.	Reflexes lost.	Obstinate constipation. Incontinence of fæces and urine.	Death from asphyxia.	The most minute (naked eye) examination failed to discover any change in the brain, spinal cord, or peripheral nerves.
At breakfast, felt difficulty of swallowing, and was unable to swallow at dinner. 3rd day.—Progressive difficulty in raising superior extremities. 6th day.—Paralysis of all limbs and death.	....	....	Death.	Bérard found nothing in brain or cord to account for death.
General feebleness, developing Jan. 10 into paralysis of lower extremities, followed by paralysis of upper extremities and face. Delirium and death.	....	Loss of appetite and diarrhœa.	Death in delirium.	Brain and cord normal. Neuritis of right sciatic nerve.
Weakness of lower extremities, quickly increasing to complete paralysis of them. Soon after, invasion in succession of upper extremities, trunk, and respiratory muscles. Dyspnœa. Death.	....	....	Death from asphyxia.	Infiltration and ulceration of Peyer's glands. No changes observed in brain or cord.
Grasp impaired, and could not stand, sit, or raise his knees in bed.	....	....	Gradual recovery, but legs feeble at end of eight weeks.	....
For two or three days difficulty in walking, and July 17, complete paralysis of all extremities, and next day paroxysms of dyspnœa, and death.	....	....	Death from asphyxia.	....
April 20.—Child could not raise head, and next day hands weak and lower extremities completely paralysed. Voice weak. Dyspnœa. Death.	....	....	Death from asphyxia.	....
Suddenly seized with difficulty in walking. Improved in few days.	....	Mental confusion.	Recovery.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
36.—Russell (Dr.)..	27	M.	I.—3 weeks. P.—10 days. C.—6 months.	Sexual excesses	Severe pain round the che and in the calves of his le Tactile sensibility unaffect
37.—Oulmont (M.) and Hayem (G.)	32	M.	I.—10 days. P.—8 days.	Carpenter. Said to have been sober. Exposure for 17 days to cold and wet.	Suffered for ten days fro formication and weakne of lower extremities.
38.—Harley (G.) and Clarke (L.)	17	M.	I.—1 month. P.—5 days.	None mentioned.	Aching of limbs for a mont March 12th, felt numbness legs and stiffness of nec Tactile sensibility normal.
39.—Bourdillat (M.)	3	—	P.—11 days.	August 23.—Attack of measles. Impetigo of head, and, week later, gangrene of lobule of right ear. Convalescence, and ulcerated surface almost completely healed. General feebleness remained.	Sensibility intact.
40.—Cuming (Dr.)	40	M.	P.—2 or 3 weeks C.—2 years.	Pork cutter.—Drinking heavily for a fortnight.	Numbness of hands. Finge cold and white as far second joints. Darting pai in lower limbs; no ans thesia.
41.—Bayer (O.)	35	M.	P.—21 days. C.—2 months.	Military man, in easy circumstances. Syphilitic infection five years previously. Suffered from various secondary and tertiary manifestations.	No paræsthesia. May 15.—Blunting of sens bility in lower extremitie Severe attack of cardialgia
42.—Lange (C.)....	—	M.	P.—5 months.	....	....
43.—Chalvet (J. W.) Kiener's case.	27	M.	P.—3 days.	Attack of smallpox, which aborted at end of a week.	Formication of fingers an toes; sensibility to pain an touch diminished.
44.—Bernhardt (M.)	29	M.	I.—6 days. P.—3 days.	Mild attack of smallpox with convalescence on eighth day.	Two days after convalescen numbness of right hand, an some loss of muscular sens Amblyopia of right eye.
45.—Reincke (J.) ..	30	M.	P.—28 days. C.—5 months.	Gardener's assistant. No syphilis and no undue exposure.	Formication in arms and leg No anæsthesia. Muscles te der on pressure. Gre hyperæsthesia of arms.
46.—Duchenne	Young	F.	P.—12 days.	None mentioned.	Formication and numbness lower, and, few days lat of upper, extremities. Dim nution of cutaneous sens bility.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
Weakness of legs since Christmas. Cramp in the calves from three weeks. Admitted August 18, and then almost completely paraplegic.	....	....	Partial recovery in 6 months.	....
October 22nd.—Mounted the Hospital stairs without help. 2nd day.—Gait uncertain. 5th day.—Could not stand, and upper extremities feeble. 8th day.—Great increase of paralysis and death from it.	....	....	Death from asphyxia.	No definite morbid changes discovered in medulla or cord on microscopic examination.
March 13.—Difficulty in walking. Fifth day lower extremities completely, and upper partially, paralysed. Dysphagia and dysarthria, dyspnœa, death.	....	....	Death from asphyxia.	Clarke found softening in the cervical region. Other parts of cord and medulla normal.
Oct. 5.—Weakness of all extremities. Nasal voice and dysphagia. Symptoms progresses, and Oct. 16.—Paralysis of diaphragm and death	....	Paralysis of sphincter towards end, and also pneumonia developed.	Death from asphyxia.	Pneumonia. Ranvier found on microscopic examination the nerve centres healthy.
Could not lift cup to lips. Second day, difficulty in walking, and third, could not stand. Ninth day, almost completely paralysed below neck. Respiration embarrassed. Tenth day, slight improvement.	Reflexes absent ..	Profuse sweat. Pulse 126.	Partial recovery in two years. *Main en griffe* persisted.	....
May 5, 1867.—Fell in mounting his horse; heaviness and helplessness of his legs. Next morning difficulty in getting up, and third morning could not leave his bed. May 15.—Lower extremities almost completely paralysed, and upper very feeble. May 26.—Improvement, which progressed to complete recovery by end of July.	....	Weakness of bladder and rectum. Numerous purpuric spots on toes.	Recovery, under anti-syphilitic treatment, in 2 months.	....
Paralysis spread gradually from lower to upper extremities, and death from respiratory paralysis at end of five months.	Reflexes diminished.	....	Death from asphyxia.	....
December 26.—Legs flexed under him. Second day, lower extremities paralysed and upper feeble. Third day, aphonia, dysphagia, dyspnœa, and death.	Reflexes lost	....	Death from asphyxia.	Nothing beyond congestion of spinal cord.
Weakness of lower extremities for six days. Seventh day, legs completely paralysed, and arms weak. Speech thick, slight dysphagia. Ninth day, respiratory paralysis.	Electrical reactions normal.	....	Death from asphyxia.	No morbid changes found on microscopical examination of medulla, cord, nerve roots, and nerve trunks.
Increasing weakness of legs, and could not stand without support at end of eleven days. Next day arms affected; diplopia. Increase of paralysis for other fifteen days, then slow improvement.	Faradic contractility normal. Reflexes lost.	Sleeplessness. Muscular atrophy.	Partial recovery on leaving hospital at end of five months.	....
Feebleness of extremities, and second day took to bed. Paralysis increased gradually, and twelth day death.	Progressive diminution of faradic contractility.	Bladder and rectum paralysed.	Death by asphyxia.	No lesion appreciable to naked eye in nervous centres.

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
47.—Gombault (M.)	35	M.	I.—4 days. P.—1 month. C. Prolonged.	None mentioned.	Numbness and formication of extremities
48.—Gombault (M.)	67	F.	P.—5 days. C.—Prolonged.	None mentioned.	Sudden numbness and heaviness of limbs. No anæsthesia.
49.—Lévy	—	M.	P.—8-16 days. 4 weeks.	Began by headache, vertigo, and vomiting. Supposed to be of rheumatic origin. (Author contributes 2 other analogous cases.)	No anæsthesia.
50.—Bernhardt (M.)	35	M.	I.—7 days. P.—7 days. C.—12 months.	Neurotic family history. Soft chancre 1862, but no constitutional symptoms. In 1866 fainting attack; 1867, pains in neck, difficulty of swallowing, and an uncertain and staggering gait, from which he made a slow but complete recovery under anti-syphilitic and hydropathic treatment. In 1871 attack of catarrhal jaundice; unfortunate in business. No mention of habits. January 1, 1872.—Attack of diarrhœa, and on night of 7th severe sweating, and went out next morning lightly clad.	Complained of pains which, on exertion, shot from the gluteal region to the head. Moderate degree of pains in calves of legs, and tenderness of various parts of the body on pressure. No numbness or tingling complained of, and no anæsthesia.
51.—Bernhardt	22	M.	P.—12 days. C.—2 months.	Student. Drinking new beer. Diarrhœa; exposure to cold.	No anæsthesia. Muscles tender to pressure.
52.—Bernhardt	20	M.	P.—2 days. C.—Slow.	Beginning of January, exposure to cold. No mention made of habits.	Muscles tender to pressure.
53.—Eisenlohr (C.).	33	M.	I.—Few hours. P.—12 days. C.—10 weeks.	Merchant. Healthy till Easter, 1873, when he had, after exposure, a painless motor weakness of both arms, which disappeared in a few days. May 30.—First symptoms began after dancing all night, and exposing himself to cold whilst heated.	May 30. — Lancinating pains in lower extremities. No paræsthesiæ and no anæsthesia.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions.	Termination.	Morbid Changes.
After four days' malaise, extremities became paralysed. At end of a month gradual improvement. Atrophy of right arm and left leg persisted.	....	Fever during first week.	Partial recovery, with persistent atrophy of some muscles.	....
Weakness and inability to stand in half an hour. Fifth day entered hospital, and found completely paralysed in limbs, with embarrassment of breathing. Gradual improvement began, but at end of 3½ months atrophy of muscles of hands and feet, with deformities, appeared and persisted.	Loss of faradic contractility in muscles of hands and extensors of fore-arms.	....	Death, seven years later, from cancer of abdomen.	Ganglion cells of anterior horns of cord were globular, and contained excess of pigment. Degenerative fibres in some of anterior roots. Islets of sclerosis, with excess of connective tissue in peripheral nerves.
Weakness of lower extremities, and in four days complete paralysis of them. 8th Day.—Paralysis of right facial, and few days later dyspnœa, and paralysis of epiglottis and œsophagus. Application of hot iron to spine. Symptoms stationary for eight days, then improvement.	Rhomboids, serrati, and infraspinati atrophied and lost their faradic contractility.	....	Recovery in four weeks.	....
January 8th, 1872.—Found he could not hold soap whilst washing; could hardly get change from his pockets, and had to be assisted up steps of his house. Second day, admitted to hospital; third, movement of hands and fingers nearly lost, and lower extremities almost completely paralysed. Increase of paralysis up to January 15; afterwards slow improvement.	Reflexes lost. Mechanical irritability of extensors increased at first. Electrical re-actions normal for eight days. Afterwards faradic contractility became progressively diminished, and finally lost in some muscles, and in them the galvanic current gave R. D.	....	At end of twelve months patient could not stand without support. Had double ankle drop, and movements of upper extremities remained feeble.	....
January 10.—Slight helplessness of right upper and lower extremities, and slight dysphagia. Second day could not raise himself without help, and ten days later great helplessness.	Electrical reactions normal.	....	Partial recovery in two months.	....
Weakness of all extremities quickly followed exposure, and complete paralysis of them a few days later. Atrophy of muscles of hands.	Diminution of faradic and galvanic contractility of affected muscles.	....	Very incomplete recovery in two months.	....
Weakness of all extremities followed these pains in a few hours, and a few days later the patient could not stand or walk. June 6.—Grasp feeble. Lower extremities nearly paralysed, the gluteal muscles and extensors of knees being more affected than muscles of legs. June 9.—Great increase of paralysis, but two days later slight improvement, which, subject to temporary relapse, continued.	Electrical reaction normal.	Œdema of ankles. Swelling and redness of joints of left hand.	Patient recovered completely.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
54.—Calestri (L.) ..	29	M.	P.—About a month. C.—18 days.	Had attack of some lung affection, and, later, acute rheumatism with cardiac complication. Fireman, and exposed to changes of temperature.	Feeling of formication and numbness in all extremities. Anæsthesia of lower extremities. *Pains along vertebral column.*
55.—Leyden (E.) ..	40	F.	P.—Two or three days.	Strong, and hitherto healthy woman.	Sudden pains in her right side. No anæsthesia.
56.—Leyden (E.) ..	60	F.	....	....	....
57.—Déjerine (J.) and Goetz	45	M.	I.—2 days. P.—3 days.	Carpenter. Contracted chancre 14 years previously. Slight sore throat, but no other secondary symptoms. Two years previously had severe headache, with diplopia, which lasted 2½ months.	Jan. 29.—Lively pains in lower extremities and arms. Deep seated pains in bones, with nocturnal exacerbation. Formication of lower extremities. Coldness of feet. No anæsthesia.
58.—Westphal (C.)	Adult.	M.	P.—28 days.	Clerk. In army, 1860, when he had gonorrhœa, with phimosis, requiring operation. Swelling in groin, but no chancre, and no secondaries. 1874.—Hæmoptysis. 1867.—Rheumatic pains in shin bone, and swelling of shins.	March 1.—Numbness of feet and toes, and subsequently of hands and fingers. No marked anæsthesia.
59.—Westphal (C.)	24	M.	I.—2 or 3 days. P.—14 days.	Coachman. Of healthy family history. Previous year intermittent fever. No infection and no alcoholism.	January 15.—Felt his hands and feet deadly cold and feet wet with sweat. Formication of hands and feet. Tenderness of muscles on pressure and on exertion.
60.—Westphal (C.)	64	M.	P.—14 days.	Had extensive ulcers on both legs. Slight enlargement of inguinal glands.	No disorder of general sensibility mentioned.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
March, 1873.—Extremities feeble, and next morning could not stand, and could scarcely move his arms. April (middle). — Improvement, which progressed, so that on May 4th he could walk.	Muscular irritability normal.	Enlargement of heart with mitral insufficiency. Enlarged spleen. May 5.—Diarrhœa, fever, and on May 8th death.	Death from acute gastro-enteritis.	Hypertrophy of heart, and signs of previous endo- and peri-carditis. Brain and cord normal to naked eye.
Weakness of all extremities; next day, dysphagia, hoarseness, and aphonia. Face drawn slightly to right. Paralysis of respiration and death.	....	Right pupil dilated.	....	On microscopic examination, small focus in medulla on right side on level with glosso-pharyngeal vagus and spinal accessory, and in part facial nerves. Rich infiltration of cells and granules near central canal.
Paralytic symptoms without characteristic course.	....	....	....	Pericentral sclerosis, with cystic softening in cervical region.
January 29.—Walked to hospital. February 1.—Complete paraplegia. February 3.—Paralysis of all limbs; intense dyspnœa, and death during night.	....	Feb. 2.—Retention of urine and paralysis of rectum	....	Spinal cord normal but medulla not examined microscopically. Degenerated fibres with multiplication of nuclei found in some anterior roots.
March 1.—Weakness of left leg, and six days later weakness in mastication. March 8.—Weakness of right leg, and now he could barely stand, and upper extremities also feeble. In evening difficulty of articulation and slight dysphagia, fluid passing through nose. Voice hoarse. March 10.—Could no longer stand. March 28.—Upper costal breathing. Dyspnœa; death.	March 11. Reflex of sole and of cremaster lost. Electrical reactions normal.	Sphincters intact. No fever. Pulse 68. Moderate swelling of inguinal glands. March 15.—Rigor. Temp. 40·4°, and afterwards of remittent type.	Death from asphyxia.	Grey tubercle scattered through both lungs. Microscopic examination of nervous centres gave negative results.
January 17.—Pains and stiffness increased, and had to go to bed. January 24.—Could hardly walk, and upper extremities weak. Gradual increase of paralysis, breathing upper costal type. Dyspnœa, and Jan. 31, death.	Electrical reactions of nerves and muscles normal.	Ronchi heard over both lungs.	Death from asphyxia.	Aorta of small calibre. Left ventricle hypertrophied. Bronchitis. Enlarged spleen. Microscopical examination of spinal cord and medulla negative.
August 29.—Sudden attack of speechlessness (aphasia?) and slight paralysis of right side of face, and inability to swallow. September 8.—Perfectly sensible, and could write replies. Muscles of both sides of face paralysed more or less. Inability to protrude tongue, and mouth could only be opened slightly. Complete inability to swallow persisted; extremities feeble. September 11. — Death from asphyxia.	Electrical reactions normal.	Cheyne-Stokes Perspiration.	Death from asphyxia.	Granular kidneys. Hypertrophy of left ventricle of heart. Liver granular. No gross lesion of brain. On microscopical examination the medulla, pons, cord, and the roots of the cranial nerves were found healthy.

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
61.—Westphal (C.)	33	M.	I.—12 days. P.—32 days.	Labourer. Of sober habits. Aug. 16.—Diphtheria; recovery. Sept. 9.—Began work, but at end of 8 days had to desist from general weakness.	Sept. 29.—Feeling of deadne in his fingers and hands, an of numbness of face. Late numbness and diminutio of the sense of pain and c touch, and loss of muscula sense in lower extremities.
62.—Baumgarten (P.)	52	M.	P.—7 days.	Cough and expectoration for three years. Three weeks before onset was rubbed on the chest for rheumatic pains by fat supposed to have been derived from a horse dead of splenic fever.	February 9.—Began suddenl with feelings of formicatio in lower extremities. Cuta neous sensibility lost t lower, and much diminishe in upper, extremities.
63.—Salomon......	24	F.	P.—2 days. C.—14 days.	Hitherto healthy.	Widely diffused anæsthesi Deep pressure of vertebr column painful, but applic tion of hot iron was not fel Anæsthesia disappeared i two days, and was replace by hyperæsthesia of low extremities.
64.—Velden (R.v.d.) and Leyden (Prof.)	52	M.	Two months. P.—3 days.	Three years previously ulcerated legs. Dec., 1876.—Ceased work from weakness caused by continuous diarrhœa, with loss of appetite. Jan. 13, 1877.—Admitted to hospital. Diarrhœa disappeared with treatment, but great weakness remained. Jan. 29.—Recurrence of diarrhœa.	January 30.—Painful creepin and formication felt sudde ly in lower extremities. N anæsthesia.
65.—Eisenlohr (C.)	42	M.	P.—9 days.	Mason. Contracted chancre 14 years previously.	No marked sensory disorder

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
September 29.—Great weakness of lower extremities. October 16.—Admitted to hospital. Speech nasal and indistinct, and immobility of soft palate; regurgitation through nose; dysphagia. Grasp feeble; claw hands, and weakness of arms; unable to walk without support. October 20.—Attack of dyspnœa, and few days later breathing of upper costal type. October 31.—Death from asphyxia.	Electrical reactions normal. Knee-jerks lost.	Pulse, 78. Resp., 20. Oct. 26.—Slight bed sore.	Death from asphyxia.	Slight pneumonia. Cerebral pia mater milky. The medulla and cord normal. On microscopic examination, transverse section of anterior crural nerve showed similar changes to those found in musculo-spiral nerve in a case of lead paralysis.
February 9.—Weakness of lower extremities. February 15.—Admitted to hospital, and on following day all extremities completely paralysed. Paralysis of diaphragm; dyspnœa; death.	Reflex irritability lost.	....	Paralysis of diaphragm, and death from asphyxia.	Micro-organisms found in the blood similar to those observed in the blood of a horse dead of splenic fever.
Gradually progressing ascending paralysis, involving muscles of neck, tongue, and face. Respiratory muscles unaffected. Improvement in two days, and complete recovery in 14 days.	....	Bladder and rectum slightly affected.	Complete recovery in 14 days.	....
January 30.—Could not undress himself. January 31.—Inability to stand, and weakness of upper extremities. Same day attack of dyspnœa. Speech like that of drunken person, but no dysphagia; sopor; death.	Electrical reactions completely lost. Reflexes lost. Mechanical irritability retained.	Temp., 37·4 to 39·3. Pulse, 62-68. Delirium January 24, with long incursion of abdomen. Cheyne-Stokes respiration. Incontinence of urine.	Slight delirium; sopor; death.	Spinal cord examined by Leyden microscopically. Swelling of axis cylinders of nerve fibres in certain foci of myelitis. Ganglion cells pigmented and swollen, and others atrophied, but did not observe multiplication of nuclei. Nerve trunks showed minor changes.
October 7.—Weakness in arms and legs, and two days later complete paralysis of all extremities. October 16.—Muscles of abdomen weak. Slight dysphagia, and uvula directed to left, and its reflex irritability sluggish; speech toneless. Dyspnœa and death.	Reflexes lost. Faradic contractility normal.	Pulse, 136. Temp., 37 C.	Death from asphyxia.	Hypertrophy of heart; atheroma; hyperæmia of kidneys. Morbid changes found in cord at level of third cervical nerves and upper lumbar region. The ganglion cells pigmented, and central canal filled with cells. In pons foci, filled with white blood corpuscles. Capillary hæmorrhages in pons and medulla.

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
66.—Jaffé (Th.)	25	M.	I.—2 days. P.—8 days.	Merchant. Contracted chancre ¾ year before onset. Roseola on breast and arms. Mercurial inunction. Venereal excess for some weeks. Attack of diarrhœa.	October 2.—Feeling of hea ness, and took to bed. anæsthesia.
67.—Kétli (K.)	—	—	....	Poisoning by corrosive sublimate.	Anæsthesia.
68.—Kélti	—	—	Some hours. P.—Short time.	Poisoning by corrosive sublimate.	Complete anæsthesia.
69.—Cornil and Lépine	20	M.	P.—3 days.	No data given.	Anæsthesia of feet and legs.
70.—Kahler (O.) and Pick (A.)	12	F.	I.—11 days. P.—11 days.	October 5.—Noted she had been fretful for 8 days, but went to school for 3 days longer.	For 11 days had feelings pressure in fingers and to October 9.—Felt severe p in hollow of knees and bu ing pains along verteb column. Later, blunting of tactile s sibility and inability to d tinguish small differences temperature, and dela sensibility to temperature lower extremities.
71.—Kahler (O.) and Pick (A.)	33	M.	P.—20 days. C.—5 months.	Butler. Venereal excesses, but not an habitual drunkard. Gonorrhœa, but no syphilis. Led an irregular life.	Feelings of coldness and num ness of feet and legs, but pain. Delayed sensibili and separation of pain and tactile impressions feet.
72.—Kümmell (H.)	25	M.	P.—6 days.	Saddler.—Attack of typhoid fever. Convalescence at end of 4 weeks. Could sit up whole day, but felt legs heavy.	No paræsthesia, pain, or oth sensory disorders.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
October 4.—Lower extremities paralysed, and next day upper extremities weak. Next three days increasing paralysis; paralysis of diaphragm; dyspnœa; death.	Reflexes lost. Electrical reactions normal at first; later, marked, diminution of faradic contractility and two days later Erb found it lost. Galvanic contractility also diminished; no R.D. Patellar-tendon reactions lost. Mechanical irritability increased.	Retention of urine.	Paralysis of diaphragm, and death from asphyxia.	....
Weakness of extremities	Diminution of reflex irritability.	Usual phenomena of sublimate poisoning.	Recovery	....
Acute ascending paralysis, running a fatal course.	Loss of reflex irritability.	Usual phenomena of sublimate poisoning.	Death	....
February 4.—Paralysis of left upper and of lower extremities. 3rd day.—Right upper extremity attacked. Soon afterwards, dyspnœa and death.	....	No fever.	Death from asphyxia.	Slight degree of softening of cord. Granular bodies and granulations scattered throughout the whole of the cord.
October 9.—Weakness of legs and next day absolute paralysis of them and weakness of arms. October 12.—Patient had to be fed. October 18.—Admitted to hospital. All the extremities paralysed, and muscles of back and abdomen weak. Fluid finds way into glottis in swallowing. October 20.—Paralysis of muscles of abdomen, dysphagia, dyspnœa, and death.	Reflexes lost. Knee-jerks lost. The nerves give normal faradic and slightly diminished galvanic reactions. No R. D. of muscles.	Phenomena similar to tâches cérébrales. Pulse 120, breathing 50.	Death from asphyxia.	Enlargement of mesenteric glands and of root of tongue. Enlarged spleen, tonsils swollen, œdema of lungs, purulent bronchitis. On microscopal examination cord found healthy.
December 16.—Noted slight weakness of lower extremities. 2nd day.—Could hardly stand. 3rd day.—Took to his bed; and on 7th day, lower extremities completely paralysed. Jan. 4.—Slight improvement, and left hospital April 12, recovered.	Reflex of sole lost. Patellar-tendon reactions lost. Electrical reactions normal.	Difficulty in urinating.	Recovery in three months.	....
Weakness of legs extended to muscles of back, and in two days patient could hardly walk or sit up. Jan. 11th.—Admitted to hospital. Weakness of legs, and insecurity in walking. Jan. 12.—Could not stand; regurgitation of fluid through nose; could not open mouth completely; articulation indistinct. Jan. 13.—Paralysis of extremities showed slight improvement; Bulbar symptoms worse; double facial paralysis. Jan. 15.—Complete paralysis of all extremities. Died in evening from œdema of lungs, and paralysis of vagus.	Electrical reactions normal.	Enlarged spleen; pulse 80, rising to 160. Temp., 36·8.	Death from œdema of lungs and paralysis of vagus.	Œdema of lungs, Peyer's patches, and solitary glands enlarged and ulcerated. Hæmorrhage about size of a pea in the restiform body on one side, and another rather less on the opposite side in the lower half of the medulla; cord normal.

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
73.—Fox (A. W.) ..	26	F.	I.—Several months. P.—8 days.	Prostitute. Syphilis six years before. Had not gone to bed sober for seven years.	Complained of great languo and loss of appetite fo several months. For a week pains in legs and loin. March 21.—Admitted to hospi tal. Hyperæsthesia an hyperalgesia of lower limb and, to a less extent, of arm Patient screams when move or touched.
74.—Schulz (R.) and Schultze (F.)	44	M.	I.—4 weeks. P.—23 days. C.—4 weeks.	Contracted syphilis the previous year.	No sensory disorders at firs Later, numbness of toes an formication of fingers. Pai in legs and loins.
75.—Hünnius (G.)	64	M.	P.—6 weeks. C.—4 months.	Symptoms began three weeks after exposure to severe cold.	Formication of toes and le No anæsthesia.
76.—Finney (J. Magee)	25	F.	P.—9-12 days.	Unmarried, and away for 2 or 3 weeks from home with male companion. On returning home, 3 days before admission, could hardly walk. No external injury and apparently no alcoholic excess, but patient very reticent.	Uncomfortable feeling do sacrum. No anæsthesia.
77.—Myrtle (A. S.)	25	F.	I.—2 months. P.—46 days.	Profuse menorrhagia. Chronic alcoholism.	Jan.—Numbness and coldn of extremities. March 10.—Excruciating pai Skin sensitive to touc Sensation as of tight co round waist. Hemianops
78.—Schultze (Prof.)	44	M.	P.—6 months.	Jan., 1882, syphilis, and in May paralysis began.	No paræsthesia or pains. anæsthesia.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
For months felt languor, and for six weeks had to pull herself up by arms in getting up. March 21. — Lower extremities completely, and upper partially, paralysed. Speech indistinct, and could not protrude tongue beyond lips. Progressive increase of paralysis, dyspnœa, and, March 29, death.	Tendon reactions lost.	Incontinence of urine. Albumen in urine. Constipation. Pulse, 160. Temp., 98°-100°-2. Resp., 32. Delirium.	Death from asphyxia.	Liver large and fatty, with gumma. No change noted in brain or cord, or in their membranes. On microscopic examination, minor and diffuse changes in spinal cord. Lumbar region quite healthy.
For 4 weeks felt legs heavy and stumbled a little. September 29.—Legs distinctly paretic. October 1.—Unable to stand, and 2 days later arms weak, slight dysphagia, and feebleness of mastication. October 5.—Could no longer swallow solid food, mastication feeble, and all extremities nearly completely paralysed, and abdominal muscles weak. October 21.—Slight improvement, which continued for 4 weeks, when bronchitis supervened. November 26.—Patient died.	Reflexes and tendon reactions lost. Faradic contractility lost and galvanic R. D.	Constipation. Pulse 90. Temperature normal. Slight incontinence of urine.	Death from bronchitis.	Soft spots found in dorsal and cervical regions of cord. On microscopical examination morbid changes were found in lateral columns and anterior horns, ganglion cells swollen and showing vacuolation. Minor changes found in peripheral nerves.
Weakness gradually increasing in 4 weeks to almost complete paralysis of lower extremities. 13 days later all extremities paralysed, and in four days more masticatory muscles feeble, articulation indistinct, and respiratory difficulties. Gradual improvement, and almost complete recovery in four months.	Reflex of sole lost. Knee-jerk lost.	....	Recovery almost complete in four months.	....
Dec. 5.—On returning home could only just stand. Dec. 8.—Admitted to hospital. Complete paralysis of lower extremities, and could hardly sit up, and two days later upper extremities invaded. Dec. 12.—Respiration carried on almost entirely by diaphragm. Dec. 13.—Dysphagia. Dec. 14.—Death from respiratory paralysis.		Constipation, and retention of urine, probably from paralysis of abdominal muscles.	Death from asphyxia. Diaphragm the last to be paralysed.	No naked eye changes in brain or spinal cord.
Jan.—Weakness of knees. Mar. 16.—Tumbling gait, and in a few days inability to move feet freely in bed. Apr. 6.—Muscles of thumb atrophied. Apr. 10.—Right eyelid drooped. Double squint, breathing deep and slow. Complete paralysis, except of the facial muscles. Apr. 30.—Death in coma.	Galvanic current caused great pain, but no contractions.	Epileptiform seizures. Semi - conscious and rambling.	Died comatose.	. . .
May.—Weakness of legs, and 14 days later difficulty of writing and in raising right arm. July 18.—Uncertain, waddling, and high-stepping gait, grasp feeble, and 14 days later both arms weak. Still later dyspnœa supervened. Nov. 14.—Death.	Electrical reactions normal except in peroneal region, where galvanic current gave A S Z > K S Z.	....	Death from asphyxia.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
79.—Schultze (Prof.)	30	M.	P.—7 months.	Farmer.—No known cause.	Pains in region of hip.
80.—Rumpf	12	M.	P.—8 days. C.—7 months.	None assigned.	Anæsthesia, and loss of muscular sense in lower extremities.
81.—Ross (J.).	21	F.	P.—5 days.	Prostitute. Syphilis. Blow on back of neck, but no external mark.	No marked sensory disorder.
82.—Ross (J.).	45	F.	P.—12 days.	No history.	Muscles tender to pressure.
83.—Ross (J.).	23	M.	P.—4 or 5 days. C.—4 weeks.	Sores on penis; sexual excess and nocturnal emissions.	Numbness in hands and fingers, and next day in feet also. No anæsthesia.
84.—Jacoby (M.) ..	28	M.	I.—2 days. P.—28 days.	Merchant. Obesity. Had to travel much, and led an irregular life.	July 21.—Pains in the thighs and heaviness of limbs. No manifest anæsthesia.
85.—Hoffmann (J.).	36	F.	I.—10 days. P.—14 days.	Washerwoman. For 8 days felt unusual lassitude whilst at work. July 16th and 17th worked hard at washing, and perspired freely, but was not exposed to cold.	July 18.—Complained of formication of thighs. No pain and no anæsthesia. Tinnitus aurium.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
May.—Increasing weakness of left leg. Sept.—Right leg also weak, and 2 weeks later left arm attacked. Oct.—Weakness of all extremities. Dec. 15.—Death from respiratory paralysis.	Electrical examination gave the partial form of R D. Patellar-tendon reactions lost on left and weak on right side.	Profuse sweat.	Death from asphyxia. Diaphragm paralysed last.	....
Quickly progressing paralysis of lower extremities, and of muscles of abdomen and back, and weakness of upper extremities. Dysphagia, dyspnœa. At end of 8 days improvement and gradual recovery.	Electrical reactions normal. Reflex of sole lost. Patellar-tendon reactions lost.	....	Complete recovery in 7 months.	....
January 26, 1887.—Admitted to hospital with complete paralysis in lower, and partial paralysis in upper, extremities. 2nd Day.—Complete paralysis of all extremities, and dyspnœa. 3rd Day.—Death.	....	....	Death from asphyxia.	Cicatrices in groin. Minor changes found in grey matter of spinal cord.
Sudden paralysis, increasing for ten days. On entering hospital, lower extremities completely paralysed, and upper feeble. 2nd Day.—Sudden death.	Patellar - tendon reactions lost. Faradic contractility normal.	....	Death from asphyxia.	Brain and spinal cord found normal on microscopic examination.
For few days loss of power. Dec. 10.—Unable to sit up or turn himself in bed, and later, same day, lower jaw dropped, dysphagia, and slight drooping of upper eyelids. Dec. 11.—Gradual improvement.	Reflex of sole present. Patellar-tendon reactions lost.	Dilatation, and inequality of pupils. Constipation.	Recovery in four weeks.	....
July 23.—Difficulty in standing and stamping gait, but could move extremities feebly on lying down. Aug. 5.—Double wrist-drop. Aug. 20.—Intercostal and accessory muscles of respiration paralysed, and breathing diaphragmatic. Death.	Faradic contractility lost. Reflexes and tendon - reactions lost.	No manifest atrophy.	Death from asphyxia. Diaphragm last to be paralysed.	....
July 17.—Heaviness in lower extremities and difficulty in ascending stairs; next day took to bed, and upper extremities weak. July 21.—Unable to stand, difficulty in mastication, voice indistinct. Few days later dysphagia and droop of upper eyelid of right side. Aug. 1.—Admitted to hospital. Unable to raise thighs in bed, but movement of feet fairly strong. Paresis of upper extremities and grasp feeble. Masseters and orbicularis oris paralysed, mouth open, and overflow of saliva. Ptosis of right eye. Aug. 2.—Increase of symptoms, paralysis of diaphragm, dyspnœa, and death.	Reflex of sole retained. Patellar-tendon reactions lost. Faradic contractility normal for nerves and muscles of extremities and left side of face, but was diminished for right facial nerve and muscles.	No atrophy of muscles. Constipation.	Diaphragm paralysed. Death from asphyxia.	Myelo-meningitis and bulbo-meningitis, but lumbar enlargement of cord was free from change. Degenerative changes found in the right facial nerve, but the other nerves were normal.

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
86.—Inmermann (Prof.)	22	M.	P.—7 days. C.—4 weeks	......	No sensory disorders.
87.—Sorgenfrey (D.)	57	M	I.—7 days. P.—4 days. C.—7 days.	Wet and exposure when body heated.	No sensory disorders be feeling of heaviness vertebral column.
88.—Mieth (H.)....	42	M.	P.—29 days. C.—5 weeks.	Factory worker.	Formication and numbne hands and feet.
89.—Curschmann..	31	M	P.—9 days.	Waiter. June 1st, seized with rigors, and on the 7th was sent to hospital.	....
90.—Möbius (P. J.)		M.	P.—24 days. C.—2 to 3 months.	Severe attack of whooping cough for six weeks, the whoop being replaced by a simple cough for some days.	No pain. Age of patient it difficult to test sensib Probable loss of tactile sibility in legs.

Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
Nov. 22.—Under feverish phenomena an ascending paralysis of the lower and upper extremities quickly developed and the muscles of abdomen and bladder were feeble. Nov. 27.—Bulbar symptoms, disappearing in a few days, and return of power to bladder and rectum. Stationary period of 4 weeks.	Electrical reactions normal.	....	At end of 4 weeks attack of croupous pneumonia and death.	Anterior horns showed spots of brick red colour. On microscopic examination single vessels found distended by red blood corpuscles. Ganglion cells filled with a hyaline mass. Granular cells scattered along walls of vessels. Peripheral nerves and muscles normal.
Lassitude and heaviness of lower extremities for 7 days. April 7.—Inability to stand, and 2 days later complete paralysis of lower, and weakness of upper, extremities. April 10.—Complete paralysis of all extremities—loss of voice, dysphagia, dysarthria, dyspnœa. April 11.—Improvement began and recovery took place in week.	Cutaneous reflexes retained. Patellar - tendon reflexes lost.	Constipation	Complete recovery.	....
July 5.—Paresis of lower extremities and of hands. July 12.—Could no longer stand and could not extend fingers; dysphagia and dysarthria. July 30.—Improvement. Aug. 12.—Could walk by aid of stick, and, Dec. 1.—Returned to work.	Patellar - tendon reactions lost, but Dec. 1, they reappeared. Electrical reactions normal.	Impotence.	Complete recovery.	....
June 7. — Admitted suffering from a quickly ascending paralysis, and 9th, death.	....	High fever.	Death from asphyxia.	Spleen 4 times normal size. Peyer's patches swollen and slightly ulcerated. On microscopical examination of cord Eisenlohr found typhoid bacilli scattered throughout the white substance and sometimes forming small masses. Bacilli cultivated and characteristic disease communicated to mice and rabbits.
As whooping cough disappeared loss of power in legs observed, and on admission Nov. 23 could not stand. Dec. 13.—Improvement of legs, but arms and muscles of neck almost completely, and the diaphragm completely, paralysed. Voice weak. Dec. 17.—Improvement: first of muscles of neck and then of diaphragm.	Reflex of sole present on left and indistinct on right side. Knee-jerks and tendon reaction of arms absent. Feb. 1.—Left knee-jerk returned.	....	Complete recovery in 2 to 3 months.	....

Observer.	Age.	Sex	Duration.	Causation.	Sensory Disorders.
91.—Mann (J. Dixon)	48	M.	I.—6 days. P.—5 days.	Hawker. No neurotic taint, syphilis, or alcoholic excess.	Jan. 17.—Cold sensation in legs, and tingling in toes and feet. Slight chills. No anæsthesia. Muscles not tender to pressure.
92.—Fére (Ch.)	41	M.	P.—6 days.	Engineer. Excessive work.	None mentioned.
93.—Bristowe and Horsley ..	—	M.	I.—2 days. P.—3 days.	Bitten by rabid cat. Symptoms began six weeks after bite.	Began with obscure symptoms, like perityphlitis. 2nd day.—Pain in back.

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Motor Disorders.	Reflexes, Tendon Reactions and Electrical Reactions.	Organic Functions and Psychical Functions.	Termination.	Morbid Changes.
January 23.—Walked with difficulty, and next day could not stand or walk. January 27.—Absolute paralysis of legs and trunk, and could hardly raise arms from bed. January 28.—Complete paralysis of extremities, trunk, and neck. No bulbar symptoms. Embarrassment of breathing; death.	Cutaneous reflexes lost. Tendon-reactions lost. Faradic and galvanic reactions normal.	Temp., 98·2° F. Pulse, 72. Œdema of ankles. Urine loaded with albumen, and solidified in test-tube when boiled. Unequal size and sluggish reaction of pupils.	Death from asphyxia.	Kidneys of normal size, but of dark colour and congested. No morbid changes found in brain or spinal cord on careful microscopic examination by Dr. Robinson. Nerves not examined.
Began by weakness of lower extremities, which next day increased to complete paralysis. Upper extremities soon invaded, and sixth day paralysis of diaphragm, and death.	Patellar - tendon reactions lost. On electrical examination muscular contractility found absent on fourth day.	Pulse 180. Temp. 37·8.	Paralysis of diaphragm, and death from asphyxia.	....
Third day paralysis began. It pursued a quickly ascending course. Death third day.	....	....	Death in 3 days.	Inoculation of rabbits with virus from the spinal cord caused rabies and death.

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Causation.—Very little is known of the predisposing causes of acute ascending paralysis. In a considerable number of the reported cases it is expressly mentioned that the patients had not inherited a neurotic disposition, but in most of the cases no particular inquiries seem to have been made to determine this question. With regard to occupation, all that can be said is that, with one or two notable exceptions, the patients have belonged to the wage-earning community, such as gardeners, butchers, masons, and carpenters, and to those who have to lead irregular lives, like commercial travellers. All that can be said in reference to the influence of the season of the year is that a considerable number of the cases have occurred about the end of December or beginning of January, and it will hereafter be shown that some of the symptoms presented by these cases give additional significance to the time of year at which they have occurred. Most of the reported cases have occurred in the male sex, the proportion in our collected cases being 62 males to 23 females, and 8 in which the sex is not mentioned. With regard to age, the majority of cases have occurred between the ages of twenty and forty years, but persons have been attacked as early as 3, and as late as 67 years of age.

In reference to the exciting causes of this disease, we cannot do better than first adduce Landry's own testimony. Although he only reports one case in detail, he states that he personally observed four other cases, and he collected from literature five cases, making ten in all. Of these, two began during convalescence from acute disease—the case reported in full after a series of febrile attacks, the last of which was probably pneumonia, and the other case after typhoid fever—two from suppression of the menses; one in a woman convalescent after confinement; one in a syphilitic subject; one probably from malaria, as it assumed an intermittent type, and was cured by quinine; two from exposure to cold, and one in the absence of any known cause. It will thus be seen that half of these cases—two post-febrile, one puerperal, one syphilitic, and one malarial—have been set up by the causes which are now known to be operative in the production of peripheral neuritis. Turning now to Chalvet's * admirable thesis, we find that he had collected 35 cases of this disease, not counting cases of the acute paralysis which occurs after diphtheria. Of these, 3 occurred after variola, 3 after typhoid fever, 2 after measles, 5 after pneumonia (in three of these there was prolonged suppuration of the ulcerated surface left by blistering), 1 from asphyxia by the inhalation of carbonic oxide gas, 1 after confinement, 1 from suppression of the lochia, 2 from suppression of the menses, 2 from sexual excesses, 1 from moral shock, 5 from exposure to cold, and 9 from unknown causes. According to this analysis, then, 13 cases have arisen during convalescence from acute febrile diseases, 2 in the puerperal state, and 1 from carbonic oxide poisoning; thus 16 out of the 35 had resulted from the same kind of causes which are known to give rise to that progressive paralysis which is believed at present to be due to peripheral neuritis. And if to these 16 cases we add the diphtheritic ones, it may be stated broadly that half of the cases collected by Chalvet own the same kind of causes as does multiple neuritis, while the remaining half may be said to have arisen from unknown causes. We say unknown causes because we cannot believe that such a disease as has been described by Landry can take its origin in such causes as suppression of the menses, sexual excess, moral shock, or even exposure to cold, in the absence of any other co-operating external cause or constitutional taint.

Of the 93 cases collected above, three (cases 20, 78, and 79) do not belong to this category, and are reserved for special consideration. Several other cases are also doubtful examples; but inasmuch as their retention will not vitiate our general conclusions regarding the etiology of the disease, we will allow them to pass at present. Of the 90 cases which remain, one (61) began during convalescence from an attack of diphtheria, five (5, 17, 18, 43, and 44) after variola, three (7, 31, and 72) after, and one (89) in the course of typhoid fever, two (16 and 39) after measles, one (90) after whooping-cough, one (25) after uncomplicated pneumonia, four (6, 19, 23, and 26) after pneumonia complicated by

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* Chalvet, *Loc. cit.* (43) p. 23.

severe treatment by means of repeated bleedings, emetics, and blisters, one (93) after the bite of a rabid cat, eight (13, 22, 41, 57, 65, 66, 74, and 81) after syphilis, one (58) in the course of tuberculosis, one (62) from supposed infection by the bacillus of splenic fever, one (14) probably from malaria, and two (1 and 12) after a normal confinement, making 32 cases which have most probably arisen as the direct or indirect result of bacillary or other organic infection. Of the remaining cases, one (35) is said to have been caused by worry and over-work in a man of gouty habit, two (49 and 54) are ascribed to rheumatism, five (40, 51, 73, 76, and 77) to alcoholic excess, one (30) to poisoning by carbonic oxide, and two (67 and 68) to poisoning by perchloride of mercury, making eleven cases, which when added to the previous thirty-two give us forty-three out of the total of ninety cases which have resulted from the causes that we now recognise as those which lead most surely to the acute forms of peripheral neuritis. Of the remaining 47 cases, two (8 and 9) are said to have arisen from suppression of the menses, five (24, 28, 36, 71, and 83) from sexual excess, and thirteen (2, 3, 4, 10, 11, 21, 32, 37, 50, 52, 53, 75, and 87) from exposure to cold, while in twenty-seven (15, 27, 29, 33, 34, 38, 42, 45, 46, 47, 48, 55, 56, 59, 60, 63, 64, 69, 70, 80, 82, 84, 85, 86, 88, 91, and 92) no cause is assigned.

We may again remark that in our opinion such circumstances as suppression of the menses, sexual excess, and exposure to cold are wholly inadequate to give rise to such a disease as Landry's paralysis in the absence of some potent co-operating factor, so that we may arrive at the broad conclusion that of the 90 cases of the disease here collected 47 have arisen from unknown causes, and 43 from the causes which are the most usual originators of multiple neuritis.

But when these records are closely scanned, reasons will be found for believing that, in many even of those cases in which careful enquiry was made into the circumstances and diseases of the patient antecedent to the onset of the paralysis, the causes assigned by the various observers are not always the real ones. The discovery of the causes of a particular disease is a very delicate and abstruse investigation, in which the most advanced methods of the present can only carry us a little way. And when the immense additions to our knowledge of those causes that have been made by experimental pathology in recent years are considered it cannot be a matter for surprise that the statements and opinions of the observers of 30, 20, or even 10 years ago, will not bear a critical examination.

With regard to the case (61), described by Westphal, as occurring during convalescence from an attack of diphtheria, we shall accept it as it stands. Various theories may be advanced to account for the manner in which the diphtheritic poison is supposed to act on the nerve tissues, but our present knowledge cannot carry us with certainty beyond the generalisation from experience expressed in the statement that the paralysis is post-diphtheritic. The same remark applies to the cases of paralysis which have arisen during convalescence from variola, typhoid fever, and measles, as well as to those which occur in the course of syphilis, tuberculosis, and the acute stage of typhoid fever, or as the result of infection by the poison of splenic fever, rabies, and malaria.

It may be supposed that the case (25) which is said to have occurred after an attack of uncomplicated pneumonia must also be left in the same category as those which arise after typhoid and other fevers. Considering that an organism—the pneumococcus—is found in the lungs and sputa in croupous pneumonia, and that, in its mode of onset and course, this disease is more allied to the specific fevers than to a local inflammation, it would not be surprising were it to be followed by a paralysis similar to that which arises after other fevers. In the above case (25), however, the disease did not pursue the usual course of croupous pneumonia; dulness of the base of the right lung persisted for some months, and the patient at the same time suffered from diarrhœa, so that there are some grounds for suspecting that the lung affection was itself only a complication of diphtheria or typhoid fever. Another supposition is that the patient was the subject of chronic alcoholism; and considering his profession and antecedents, it is not at all an unlikely one. He was a captain of infantry, who had probably been on foreign service, for it is stated that two years previously he had suffered from acute hepatitis and yellow fever; and it is well known that soldiers on foreign service, especially those of thirty years ago, were not noted for their sobriety. It is also stated that the patient suffered from cramps of the lower extremities, and this symptom is decidedly more characteristic of the paralysis caused by alcoholic excess, than it is of that following diphtheria and other fevers. But be that as it may, the considerations just advanced amply prove that there is not sufficient warrant for the conclusion that a causal connection exists in this case between the attack of pneumonia and the paralysis which followed it, such as is justly assumed to exist between an attack of diphtheria and the paralysis which is such a frequent sequel to it. And when the cases of pneumonia, with complication, come to be closely scanned, the evidence in favour of such a connection is still weaker, although it may not be possible to give to the supposition an absolute negative. In the case (19) reported by Macario, the patient had an attack of pneumonia of the right lung, which was treated by repeated emetics and a large blister. When it is considered that the tartrate of antimony is the emetic usually employed in the treatment of pneumonia, and that it may very probably have the power of causing a diffused paralysis, such as follows poisoning by arsenic, mercury, and other metals, this circumstance alone would tend to cast a doubt upon the opinion that the paralysis was the result of the pneumonia alone.

And when the phenomena which are said to have followed the application of the blisters are taken into account still further doubt is cast upon the truth of the pneumonic theory. The patient recovered from the pneumonia, but the blistered surface, instead of healing, continued to suppurate, and became covered by a white exudation. The patient remained feeble and ill for several weeks, and as he was showing signs of improvement he was attacked by numbness and formication of the extremities, and soon afterwards by paralysis of them. At the end of another two or three weeks improvement began and advanced to complete recovery in about three months. It is almost certain to my mind that the blistered surface had got inoculated with diphtheritic poison, and that the case must be regarded as an example of a moderate degree of post-diphtheritic paralysis. Similar cases to the above have been reported by Gubler (23) and by Leudet (26). In Gubler's case the blistered surface continued to suppurate for a long time, but no mention is made of its being covered by a membrane; while in Leudet's case the blistered surface ulcerated and remained open for a long time, but it is expressly stated that it was not covered by a membrane, and it may therefore be said that in these cases there is no evidence of inoculation by the poison of diphtheria. But even if this be granted, it is less likely that the paralysis had been caused by the attack of pneumonia, than that it was a sequel to some form of septicæmia which had taken its origin in these large ulcerated and suppurating surfaces; so that no violence will be done to the facts if we place these two cases in the category of the paralyses which arise during convalescence from diphtheria and allied diseases. And this opinion is neither revolutionary nor new. Judging from Gubler's* comments it would indeed appear that Leudet himself was inclined to believe that in his case the paralysis owed its origin, not to the attack of pneumonia, but to the accidental inoculation of the blistered surface by the poison of diphtheria. This view was strenuously combated by Gubler, but he was hardly in a position to have formed an unbiassed judgment upon the point, inasmuch as he desired to sustain the theory that this kind of paralysis may arise as a sequel to a large number of acute diseases. It will, however, be immediately shown that Gubler himself was fully convinced that the paralyses which occur during convalescence from active disease, whether it be pneumonia, variola, typhoid fever, or diphtheria, are essentially identical in their clinical histories and pathology. This opinion concedes almost all that is here contended for.

Let us now turn our attention to the case (6) fully reported by

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* Gubler. *Loc. cit.*, Vol. II., 1860, p. 725.

Landry. This case has been regarded by all subsequent observers as the ideal or type to which every case which can claim to be regarded as an acute ascending paralysis ought to conform, and it consequently occupies such an important historical position as to justify us in giving here a tolerably full abstract of it.

Landry's Cases.—A pavior, aged 43 years, entered the Hospital Beaujon, under M. Gubler, on June 1st, 1859. He had suffered from some kind of intermittent fever in childhood, and had an attack of articular rheumatism at the age of 15 years, but subsequently he enjoyed fair health up to about 12 months before his admission. In July, 1858, he was laid up for some weeks in bed by fever, which was ushered in by a rigor, but he made an excellent recovery, and returned to his work. Three months later he had another rigor, followed by fever and vague pains in the extremities, which lasted three weeks, and again he made a good recovery and returned to his work. At the beginning of 1859 he suffered from slight difficulty of deglutition, and a teasing cough, but there was no fever or pain, and in the following March he was attacked afresh by rigors and fever, which was this time accompanied by pain in the side and free expectoration. The medical man who attended him said he had a "fluxion of the chest," and for treatment, he bled him three times, administered emetics, blistered his side, and put him on a low diet. From this illness he made only a very imperfect recovery, but at the end of two months he returned to his work. A few days afterwards he felt tingling sensations in the tips of the fingers and toes, and at the end of a week he was compelled to leave off work altogether, owing to the great general weakness from which he suffered. A fortnight later, June 1st, he entered the hospital. He walked a considerable distance to get to the hospital, and did not complain of any definite symptoms beyond general weakness, until nearly a fortnight after his admission. On June 13th the patient complained that in walking his knees gave way under him. On the following day sudden flexion at the knees whilst walking became more frequent; his feet felt heavy, and seemed as if "they were glued to the ground." For some days the tingling sensations which he had formerly experienced began to extend, and they gradually invaded the feet, the legs, and ultimately the thighs. In the superior extremities these sensations ascended as high as the arm, and during this ascent the forearm felt as if it were surrounded by a tight bracelet, whilst the part below this line seemed as if it were benumbed by cold. On the 14th and subsequent days walking became more and more difficult, and on the 17th the patient was unable to raise his feet, and on making a few steps they trailed on the ground, but were never projected forwards in a disorderly manner. When lying on his back in bed he was unable to raise either lower extremity from the mattress, and experienced great difficulty in flexing the thigh slightly on the pelvis. In the effort to turn on his side he could move the trunk, but could not draw the legs after it. In the upper extremities he could still grasp a little, but could neither raise his arms to the horizontal position, nor maintain them in that position when raised by the observer. He complained of a feeling of stiffness in his fingers, and they felt, when he tried to move them, as if surrounded by a bandage. There was no fever, no pain either in the limbs or along the vertebral column, no headache, and no contractures or convulsions. There was some degree of blunting of sensibility in the soles of the feet. The intelligence was clear. The reflex reactions were lost. The visceral functions were normally performed, although the appetite was not very good.

June 20th.—The lower extremities were almost completely paralysed, and although the movements of the upper extremities were not entirely lost, the patient was unable to make use of his hands for any purpose. The tingling which was formerly localised in the distal segments of the extremities was now felt around the thorax and at the base of the neck. The patient also complained of a painful constriction round the chest, and on examining the chest, it was found that breathing was carried on by an elevation movement, whilst the movement of lateral expansion was lost. The epigastrium fell in slightly during inspiration, and was protruded during expiration. The patient's speech was somewhat broken, and his cough was wanting in energy.

June 25th. The muscles of both lower and upper extremities were almost completely paralysed, and the patient was unable to maintain a sitting posture without support. The abdominal muscles still contracted feebly, the intercostal muscles were paralysed, and the diaphragm was now manifestly implicated, respiration being carried on chiefly by the action of the cervical muscles. Respiration was frequent, and there was marked dyspnœa; the voice was broken and feeble, and the cough had so little energy that expectoration was next to impossible. The masticatory muscles were feeble, the tongue could hardly be protruded, and articulation was thick, but the muscles of the face and eyeballs were not appreciably affected, although the patient complained of tingling and stiffness of the cheek. The paralysed muscles and nerves still reacted readily to the electrical (faradic) current. The reflex reactions were completely lost, but there was no retraction of the tendons, no contracture, and no convulsions. The sense of pain and of temperature and the electrical sensibility were not affected, but the muscular sense was lost in the feet and toes, and tactile sensibility was diminished in the distal segments of the limbs. The patient complained of a feeling of torpidity and numbness in the limbs which he compared to the effect of severe cold, and he also stated that his limbs were always very cold. The limbs felt likewise cold to objective examination, and the feet felt to the observer's hand at a cadaveric temperature. The patient died suddenly in the afternoon as he was being propped up, by his own desire, to have some food.

Autopsy.—The results were chiefly negative. Strong adhesions were found between the visceral and costal pleuræ on both sides, and portions of the lungs were abnormally airless and friable, but there were no tubercles. The most careful microscopic examination of the spinal cord by Bourguignon, Gubler, Robin, and Landry failed to detect any evidence of morbid change either in the grey or white substance of the spinal cord. The nerve trunks were not examined.

In this case again is encountered the question whether the paralysis is to be regarded as having been caused by the pneumonia itself or by a diphtheritic or other septic poison absorbed through the ulcerated surface left by the blister. For our own part we have no hesitation in adopting the latter of the two views. Pneumonia is a comparatively common disease, and yet out of the thousands of cases which must be observed annually all over the country, no mention is now made of paralysis as following it, while, in striking contrast to it stands diphtheria, inasmuch as in every epidemic a very considerable proportion of those attacked suffer afterwards from more or less of paralysis. Now, although the above case was reported by Landry, yet the patient was under the care of Gubler,[*] and he added a few words of commentary to Landry's report, and his views of the nature and clinical affinities of the affection are so important as to deserve to be quoted. "I would ask," he says, "if our case of extenso-progressive paralysis is not closely allied to the paralysis occurring after diphtheria, which has been specially described by Bretonneau, Trousseau, Laségue, Maignault, etc., and of which new examples are being recorded every day? Does this last paralysis appertain exclusively to diphtheria? Are the symptoms absolutely pathognomonic, or is it at least the appanage of septic diseases? By no means. I have lately seen great general feebleness arise during convalescence from an ordinary follicular sore throat—guttural herpes. . . . The kind of paralysis which follows diphtheria is thus a secondary effect of a great variety of diseases which have the effect of exhausting the nervous system, of impoverishing the constitution, and of lowering the level of the organic forces, all of these being conditions favourable for the production of permanent disorders of innervation." Although we are not inclined to adopt the whole of Gubler's views, yet it is important to notice that both he and the other instructed physicians who had watched the course of the paralysis in the case reported by Landry, recognised the identity of its clinical features with those of diphtheritic paralysis, and that they were fully convinced that the two diseases owned one common pathology. If we now transfer the four cases of paralysis after pneumonia with complication to the group of diphtheritic or septicæmic paralysis—for our present knowledge does not enable us to draw a distinct line of division between the two—and the case of uncomplicated pneumonia to the alcoholic variety, pneumonia disappears, as we believe it ought to do, from our list of causes of this disease. Whooping-cough is another disease which ought not to be admitted into the list of causes of this form of paralysis on the strength of the single case (90) recorded by Möbius. It is less likely that such a common disease as whooping cough should give rise to paralysis in one exceptional instance, than that the attack, which was an unusually protracted and severe one, should have been complicated by a diphtheritic sore throat, the presence of which had been overlooked. In epidemics of diphtheria it is not uncommon to find, as will be hereafter shown, that persons who have not suffered from sore throat or other symptoms of the disease, may be attacked by a paralysis, which conforms in every respect to the diphtheritic variety, just as in epidemics of scarlet fever, persons who have not suffered from any of the symptoms of the disease may be attacked by acute disquamative nephritis. And if an attack of diphtheria may assume a latent form in an otherwise healthy person, much more is it likely to pass unobserved when it accompanies a disease having such marked symptoms of its own as whooping-cough. It is, indeed, very likely that many of the cases here included amongst those which have arisen from unknown causes, really owe their origin to a latent attack of diphtheria, and in two of these cases certain circumstances are mentioned in the reports which add greatly to the probability of the truth of this supposition. In the case (34) reported by Gru, a weakly child suffered from rigors, which seemed to mark the onset of some severe disease, but nothing remarkable was observed until twelve days later, when the first indications of paralysis appeared. It is very improbable that there was any direct connection between the rigors and the subsequent paralysis, and the most reasonable supposition is that the child had in the interval passed through a slight and unobserved attack of diphtheria. In one of the cases (70) reported by Kahler and Pick, a girl, aged 12 years, was noticed to be fretful for about eight days, and for another eleven days she suffered from paræsthesiæ of the fingers and toes, at the end of which time the first symptoms of paralysis appeared, and proved fatal in eleven days. At the autopsy the tonsils, the glands at the root of the tongue, and the mesenteric glands were found enlarged. It appears to me that no rational interpretation can be given of the symptoms observed during the onset and course of the disease in this case, and the morbid changes found at the autopsy, except on the supposition that the patient had passed through a mild attack of diphtheria.

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[*] See Landry, *Loc. cit.*, p. 488.

With regard to normal confinement as a cause of paralysis, it is more likely that the paralysis has been preceded by some slight degree of septicæmia, than that it should result directly from the normal act of parturition. In a case of diffused paralysis, occurring three weeks after a normal confinement, which came recently under my own observation, and which will be hereafter described in greater detail, nothing could be discovered in the condition of the patient or in her circumstances, including the sanitary arrangements, which could account for the paralysis, beyond the fact that she was a very rheumatic subject. At the onset of the paralysis the skin was bathed in a sour smelling and acid sweat, and there was a slight and transient swelling of some of the small joints of the hands, but there was no elevation of temperature and the pulse remained of normal frequency. Even in this case a latent septicæmia cannot be quite excluded, but, in the meantime, we shall leave the puerperal state in our list of causes of acute ascending paralysis, merely adding, that it acts, most probably, only in association with a co-operating factor like septicæmia or some constitutional taint.

The cases which are ascribed to poisoning by malaria, perchloride of mercury, and carbon monoxide, we shall leave as they stand in the tables, merely remarking that if due consideration be given to the powerful activity of the carbon gas as a poison, even in small quantities, to its wide diffusion in nature, and to the ease with which it finds access to the organism, it is very probable that it plays a more important part in the production of acute paralysis than has hitherto been supposed.

Rheumatism and gout are known to give rise frequently to a subacute and chronic paralysis, which is at the present attributed to a peripheral neuritis, and there are no good grounds for doubting that these poisons might, under certain circumstances, contribute to the production of acute ascending paralysis. But before admitting the presence of rheumatism or gout in any one case, it is very desirable that the evidence upon which the diagnosis is founded should be carefully scanned, for in the recognition of no other disease is an error so likely to be committed as in these. Hardly ever does a case of pachymeningitis, locomotor ataxia, chronic myelitis, peripheral neuritis, especially the alcoholic variety, or tertiary syphilis come under our observation, but the patient, and, unfortunately, often also his medical attendant, has been labouring under the impression that the accompanying neuralgiform pains have been of rheumatic origin, and if the pain is found anywhere below, or even near, the ankle, it is held that the presence of gout is no longer to be disputed. When, for example, a man suffering from chronic disease of the bladder complains of an associated pain in the heel or sole of the foot, it is immediately assumed that the cause of it is a gout, of which the poison has not had sufficient activity to attain to its favourite site in the big toe; and when a patient suffers from numbness, tingling or shooting pains in the extremities as a result of over-eating and over-drinking, he is supposed to be suffering from that most mythical of all diseases, suppressed gout. And after all, in searching for the causes of this disease the question to be determined is not so much whether the patient has a rheumatic or gouty constitution, but what are the co-operating circumstances which have helped to develop acute paralysis in a person having such a constitutional taint. To adopt the usual phraseology, rheumatism and gout must be regarded as predisposing causes, but at present we want to ascertain the exciting causes.

Bearing these remarks in mind, let us now examine the cases which are said to have been caused by rheumatism and gout. In the case (49) reported by Lévy, the paralysis is said to have been ushered in by headache, vertigo, and vomiting, which the author conjectures to have been of rheumatic origin, but on what grounds he makes this supposition is not apparent. These symptoms are much more similar to those which accompany the onset of an acute disease like diphtheria than to rheumatism, and the course pursued by the subsequent paralysis is by no means unlike that of the diphtheritic variety. It must be admitted that the report of the case is much too meagre to enable us to arrive at any certain conclusion with regard to the nature of the cause; but it may be safely asserted that there is no evidence to show that it resulted from rheumatism. In the case (54) reported by Calestri the patient had previously suffered from an attack of rheumatic fever and endocarditis, which left behind an insufficiency of the mitral valves. The patient was a fireman, who was exposed to changes of weather, and the paralysis was accompanied, in addition to the usual paræsthesiæ, by pains along the vertebral column. Now pain over the spinal column itself is not a symptom of acute ascending paralysis; but in alcoholic paralysis the muscular masses on each side of it are so tender that the pain caused by slight pressure over them is easily mistaken for spinal tenderness, and we strongly suspect that this was a case of alcoholic paralysis in a man who was suffering from cardiac disease, this being a combination by no means unusual. With regard to these two cases, then, it may be safely asserted that they do not afford any evidence whatever that there is a direct connection between rheumatism and acute ascending paralysis. Now, although there are grounds for believing that the first of these cases was an example of diphtheritic, and the second of alcoholic paralysis, yet we prefer not to put any great reliance upon such meagre reports, and will therefore place these cases in the category of those which have arisen in the absence of any known cause. In the one case (35) in which the paralysis is attributed to a gouty taint, the patient, a gentleman, aged 60 years, is said to have suffered from mental worry and bodily over exertion. The paralysis, which was slight in degree, and only lasted a few days, was ushered in by severe pains and dragging in the loins, and the onset was accompanied by mental confusion. There is a possibility that in this case the temporary paralysis was due to a slight cerebral seizure, but if this view be set on one side, then alcoholic excess is the next most likely cause of it. How frequently do we find that when men have to go through an unusual amount of mental worry and bodily exertion they have recourse for aid to alcoholic stimulants, which have the double effect of blunting the sensibilities of the mind and giving a fictitious feeling of strength. Be this as it may, the evidence in favour of the opinion that there was any direct connection between gout and the paralysis in this case is so very slight that we shall place it also in the category of the cases which have arisen in the absence of any ascertained cause.

In considering the cases which have been attributed to syphilis, it must be remembered that syphilitic subjects are exposed, like the general community, to the other conditions which originate ascending paralysis, and it must not, therefore, be too hastily inferred that the antecedent syphilis is in all cases the real cause of the affection. In the case (81) reported by myself, for example, there can be no doubt that the patient had suffered from syphilis; but, like other women of her class, she had also consumed large quantities of ardent spirits, and it is quite likely that the paralysis was the effect of the latter rather than of the former poison. The same remark applies to the case (58) reported by Westphal, in which the paralysis occurred in the course of tuberculosis. I have observed two or three cases of alcoholic paralysis in persons who were the subjects of chronic phthisis, and I see no reason to doubt that an acute ascending paralysis may be set up by alcoholic excesses in such subjects. With these remarks we shall leave the cases which are attributed to syphilis and tubercle as they stand in the tables.

Turning now to the abuse of alcohol as a cause of acute ascending paralysis, we find, as already mentioned, that the disease is expressly ascribed to this agent in five cases, and considering all that we now know of the activity of this poison in causing sub-acute and chronic forms of paralysis, there are no good grounds for doubting that it was the active cause in all the acute cases that are ascribed to it. It is, indeed, highly probable that we may go a good deal further than this, and surmise that alcohol was the active cause of the paralysis in many of the cases attributed to such an insufficient cause as exposure to cold, or as sexual excess, more especially when, as in the case (28) reported by Bablon, the offence is committed with such disgusting accessories as to show that the patient had for the time lost all his self-respect. In passing in review the cases in which no adequate cause is mentioned in the reports, it will be held that probability is given to the supposition that the paralysis has resulted from alcoholic excess, provided one or more of the following circumstances are present; these are—(1) That the occupation of the patient exposes him to the temptation of frequent drinking. (2) That the patient has led an irregular life in other ways. (3) That he has suffered from profuse sweats and diarrhœa. (4) That he has suffered from a previous attack, from which he had made a partial or complete recovery. (5) That the attack had begun at the end of December or beginning of January—the festive season. (6) That in the description of the symptoms, mention is made of the presence of tenderness of the muscles, elicited either on pressure over them, or on voluntary exertion. (7) That "cramps" are one of the symptoms. (8) That in the progress of the paralysis, bulbar symptoms are either absent or occupy a subordinate position. And (9) that the paralysis has pursued a comparatively favourable course. Our justification for applying some or all of these circumstances as tests in favour of alcohol as a cause of this disease might very well be challenged, but as it is not here pretended that anything more than a certain degree of probability—slight in some, and somewhat strong in other cases —can be made out in favour of this agent, we do not deem it necessary to enter upon a defence of these tests. For after all, the question of whether alcoholic excess is or is not a frequent cause of acute ascending paralysis can only be settled by fresh observations, and some good will have been done if this discussion will have directed the attention of subsequent observers to the absolute necessity there is for subjecting the habits and antecedents of the patient to the closest scrutiny.

But now to proceed with a review of the cases themselves. In Walford's first case (3) the patient slept in an out-house in wet clothes, a circumstance which at least suggests that he had led an irregular and vagrant life, even if it does not prove that he had been drinking to excess. When two days afterwards the paralytic symptoms appeared he suffered from "fidgets," which is a muscular paræsthesia frequently met with in alcoholic subjects. Mention is made of difficulty of articulation and whispering voice, but bulbar symptoms did not form a prominent feature of the case. In Walford's second case (4), two (10 and 11) of Landry's, and one (21) of Kussmaul's cases, all of which are attributed to exposure to cold, the reports are too meagre to enable us to come to any probable conclusion as to their cause, and we shall pass them over. Of the cases (24 and 25) reported by Gomes do Valle, there is nothing in the circumstances or symptoms of the first which will enable us to form even a guess as to the nature of its cause, while in the second double pneumonia is the one assigned, but, as has already been pointed out, an alcoholic complication is by no means improbable. In

Bablon's case (28), the very grossness of the act to which the paralysis is attributed would lead us to expect that the patient had been drinking to excess at the same time, but as none of the symptoms noted are characteristic of alcoholic paralysis, it would be unsafe to come to any definite conclusion. In the case (32) reported by Handfield Jones the patient is said to have been exposed to a chill when perspiring freely. He suffered from numbness and paræsthesiæ of the fingers and toes, symptoms which are almost always present in alcoholic paralysis, although they are not peculiar to it; there were no bulbar symptoms, and the paralysis pursued a very favourable course, the legs only remaining feeble at the end of eight weeks. This case looks very like a moderate degree of alcoholic paralysis.

In Caussin's case (33) the statement that pressure in the region of the vertebral column was painful shows most probably that the muscles were tender, but the other symptoms described do not help us to a conclusion. Of the two cases (35 and 36) reported by Russell, the first, said to have been caused by a gouty constitution, has already been considered and placed amongst the cases which arose from unknown causes. The second case the observer attributes to sexual excess, but the facts that although the paralytic attack began in July the patient's lower extremities were feeble since the previous Christmas, that he suffered greatly from cramps in the calves, that there were no bulbar symptoms, and that partial recovery took place in six months, point strongly to alcohol as a cause. The next case we notice is the one (45) reported by Reincke. There is nothing definite to guide us in his occupation or what is told of his habits, but the muscles were tender to pressure and there was great hyperæsthesia of the arms; there were no marked bulbar symptoms, although diplopia is mentioned; and the paralysis ended in recovery in five months. The three cases (50, 51, 52) reported by Bernhardt are probably all of them due to alcoholic excesses. The second case (51) was avowedly the result of excessive drinking of beer, and it is already included amongst the alcoholic cases. In the first case (50) a merchant who had been unfortunate in business had suffered five years previously from an attack of difficulty of swallowing, accompanied by an uncertain and staggering gait, from which he recovered, and four years later he had an attack of catarrhal jaundice. On January 1st he was seized with copious diarrhœa, and a week later he had perspired profusely during the night and went out next morning lightly clad, and to this act of imprudence the paralysis is attributed. But surely the history of alcoholic excesses is clearly written in this catalogue of his ailments. When the paralysis began the patient suffered from pains in the calves of his legs and from tenderness of various parts of the body on pressure, no bulbar symptoms were noted, and, the degree of paralysis being very severe, the patient made only a partial recovery in twelve months. In Bernhardt's third case (52) the attack is said to have begun in the beginning of January, after exposure to cold, but no mention is made of the patient's habits. The muscles were tender to pressure, no bulbar symptoms were noted, and the patient made a partial recovery in two months.

In Eisenlohr's case (53) the patient suffered a few years previously from a painless motor weakness of both arms, from which he made a good recovery, and his last attack is said to have been caused by exposure to cold whilst the body was over-heated after dancing all night. The attack was ushered in by lancinating pains in the lower extremities, but no mention is made of muscular tenderness; bulbar symptoms were absent, and the patient made a complete recovery in ten weeks. In one (59) of the cases reported by Westphal it is expressly mentioned that there was no alcoholism, but nevertheless the symptoms are by no means unlike those of alcoholic paralysis. The attack began about the middle of January. The muscles were tender to pressure, and on exertion. Bulbar symptoms were absent, and the patient died from a complication of bronchitis. The most notable symptoms which preceded the paralysis in van der Velden's case (64) were diarrhœa and great weakness of the extremities, symptoms which are by no means unlike those caused by chronic alcoholism, and the fact that the attack occurred in January points to the same cause. But inasmuch as the paralysis was accompanied by fever—the temperature varying from 99·3° to 102·7°F.—and the patient died in delirium, whilst the most characteristic symptoms of alcoholic paralysis, like muscular tenderness, are not mentioned, it would be unsafe to pass a judgment regarding the cause in this case, and we shall consequently pass it over. In Kahler and Pick's second case (71) the patient was a butler, who committed sexual excesses, and led an irregular life in other ways; and although it is stated that he was not an habitual drunkard, yet this is quite compatible with his having indulged in occasional heavy excesses. The attack of paralysis began in the middle of December by sensations of coldness and numbness of the lower extremities, symptoms which are almost invariably present in alcoholic paralysis, although not peculiar to it, the lower extremities are alone mentioned as having been paralysed, and the patient made a complete recovery in three months. In Finney's case (76) an unmarried woman left her home in company with a male companion, and on returning three weeks later she could hardly stand. Considering the circumstances under which this woman had left her home, and the large quantities of raw spirits habitually used by the peasants and working classes generally of Ireland, it might at once be inferred that this was a clear example of alcoholic paralysis, even although the observer states that apparently there was no alcoholic excess. It must, however, be confessed that none of the symptoms most characteristic of alcoholic paralysis are mentioned, that difficulty of deglutition formed a prominent feature of the case, and that the paralysis pursued an unusually rapid course for an alcoholic affection. It is not likely that this woman would have lived, during her absence from home, in the midst of typically sanitary conditions, and it is quite possible that she may have been exposed to the poison of diphtheria, and that she returned home to die of a post-diphtheritic paralysis, to which the symptoms observed appear to me to correspond more closely than they do to the alcoholic variety. This case will, consequently, be passed over, as having probably not been caused by excessive drinking. Of the three cases (81, 82, 83) recorded by myself, the last two, and probably also the first, were to my mind undoubted examples of alcoholic paralysis. The case of the first patient has already been considered, and although it is left amongst the cases of syphilitic origin, yet the known habits of her degraded class render it quite as likely to have been the result of alcoholic excesses. The second case (82) I only saw once, and no history of her antecedents could be ascertained, but the flaccidity of her lower extremities, her dropped ankles and toes, and her loud outcry on the slightest pressure being made on the muscular masses, or on passive movements being communicated to her limbs, are so indelibly impressed upon my mind that I have as little hesitation in pronouncing the case to have been alcoholic paralysis as if I were examining the patient at this moment. In my third case (83) the patient had, before his attack, led a very irregular life, and he had contracted sores on the penis, but which were not considered to have been of the nature of true chancre by so skilful an observer as Mr. Whitehead, whose patient he had been for many years. Knowing the subtlety of the syphilitic poison, however, I was myself inclined to doubt the correctness of this opinion, and I must confess that at the time I regarded the paralysis as being of syphilitic origin. But the patient has since got married, and is leading a steady and temperate life. He is the father of several healthy children without a trace of constitutional taint, and on scanning the report of the case now, supplemented as it is by my recollections of the symptoms, I have no hesitation in pronouncing it to have been a clear example of alcoholic paralysis. In the case (84) reported by Jacoby, the patient was a merchant who is said to have had to travel much, to have become obese, and to have led an irregular life. The sensory symptoms noted are often present, although they are not peculiar to alcoholic poisoning, and although the paralysis terminated fatally, yet it pursued a comparatively chronic course, and there were no bulbar symptoms. In this case, therefore, the most probable surmise is that it owed its origin to alcoholic excesses. In the case (91) recorded by my friend, Dr. Dixon Mann, the patient was a hawker. Now, during a ten years' work in the large out-patients' department of the Royal Infirmary, hawkers, both male and female, have come under my observation and treatment by the score, and I do not think I am doing any injustice to the class in saying that I have never known one of them to have been of sober habits, certainly I cannot recall one to my recollection at present. In Dr. Mann's case the attack began about the middle of January, the patient complained of cold sensations in the legs, symptoms which are more characteristic of alcoholic paralysis than of almost any other form of peripheral neuritis, and there were no bulbar symptoms. Œdema of the ankles and albuminous urine were present, and these are frequent accompaniments of chronic alcoholism. Opposed to this view are the facts that it is expressly stated that there was no alcoholic excess, showing that strict enquiry had been made into the man's habits; that the muscles were not tender to pressure; and that the paralysis pursued a rapidly fatal course. A still stronger objection to the alcoholic theory in this case is that Dr. Mann, in whose judgment and knowledge respecting such a point, I have the utmost possible confidence, still maintains that the paralysis in his patient was not caused by excessive drinking. The case, therefore, must be left amongst those which have arisen from unascertained causes.

To sum up the result of our analysis, then eight cases (6, 19, 23, 26, 34, 61, 70, 90,) can, with considerable probability, be attributed to the poison of diphtheria or septicæmia, for it is difficult to separate the two; and seventeen cases (25, 32, 33, 36, 40, 45, 50, 51, 52, 53, 59, 71, 73, 76, 77, 81, and 82,) to alcoholic excesses, making together, twenty-five cases. Pneumonia and whooping-cough, as well as rheumatism and gout, now disappear from our list, although it is admitted that the last two poisons may act as pre-disposing causes. Leaving the cases which arise from variola (5 cases), typhoid fever (4 cases), measles (2 cases), syphilis (8 cases), tubercle (1 case), the puerperal state (2 cases), malaria (1 case), splenic fever (1 case), rabies (1 case), perchloride of mercury (2 cases), and carbon monoxide (1 case)—28 in number—as they stand in the tables, and adding them to the 25 which have been caused by diphtheria, septicæmia, and alcohol, we get 53 out of the 90 total cases as having resulted from the most usual causes of peripheral neuritis, whilst the remaining 37 cases may be grouped together as having resulted from unascertained causes.

Symptoms.—The clinical course of acute ascending paralysis may be divided into three stages, which, although not distinctly marked off from one another, are yet useful for the purposes of accurate description. These are: (1) The period of invasion. (2) The period of advancing paralysis; and (3) The period of regressing paralysis, or of convalescence in the cases which survive.

(1) *Invasion.*—Premonitory symptoms, consisting chiefly of different sensory disorders, may precede the full development of paralytic phenomena for a varying period of a few hours to several months. A stage of invasion was present in 27 out of the 90 cases here collected, and in the remaining 63 cases, essentially the same symptoms as those of the invasion period were noted, but as they were accompanied or promptly followed by paralysis, they cannot be regarded as premonitory of the disease. The most frequent symptoms observed in the initial stage are certain disorders of the cutaneous sensibility, such as numbness, tingling, formication, and other paræsthesiæ, affecting chiefly the tips of the fingers and toes, and the peripheral segments of the extremities generally. Actual pain, however, is sometimes present. It may appear as a diffused aching of the limb, or as an acute and lancinating pain shooting along the course of the chief sensory nerve-trunks of the extremities, or as localised over one large trunk, like the sciatic nerve, which is then found to be tender to pressure. Shooting pains may also be felt in the loins, or passing as a girdle round the body. In one case (68), caused by acute poisoning with perchloride of mercury, anæsthesia of the extremities was noted some hours before the onset of the paralysis. Vaso-motor and secretory disorders are present in the form of "deadness," or the condition named by Raynaud "local asphyxia," of the fingers and hands, which are then deadly cold, and covered with a clammy sweat. Another group of symptoms which form a prominent feature of the disease even at this early period, are the feelings which always accompany muscular weakness, such as great fatigue on slight exertion, langour, and heaviness of the limbs. The presence of these symptoms renders it probable that the stage of paralysis had already commenced, although the loss of motor power had not been distinguished from the general weakness which accompanies the onset of most acute diseases. Other symptoms met with, and which must most probably be attributed to irritation of the nerves of muscular sensibility, are pains caused by the slightest active or passive movements of the body, pains and tenderness of the muscular masses, and especially of those of the calves, pains shooting from the gluteal region to the head, and heaviness of the vertebral column. To the same class of symptoms also belong such phenomena as fidgets, great restlessness, and stiffness of the muscles of the neck, which are occasionally mentioned amongst the premonitory signs of the disease. The loss of appetite and general malaise which usually accompany the onset of acute diseases are also mentioned, but there is no fever, and beyond fretfulness, observed in a girl (Case 70) who had probably suffered from a slight attack of diphtheria, no psychical disorders have been recorded.

The duration of the premonitory symptoms varied, as already mentioned, from a few hours to several months. In 18 cases out of the 27 in which these symptoms had been noted the period of invasion lasted only from a few hours to 12 days, and as there is nothing in the duration of the first stage in these cases to separate them from ordinary cases of post-febrile and other forms of acute multiple neuritis they do not demand further consideration at present. The remaining nine cases, in which the premonitory symptoms lasted from 17 days to several months may profitably be subjected to further analysis. In Landry's case (6), which we have supposed to have been caused by the poison of diphtheria or septicæmia, the premonitory symptoms are said to have lasted six weeks, an unusually long period for such an acute case. A reference to the report of the case in previous pages will show that the patient had suffered from a severe attack of pneumonia, for which he was treated by emetics, bleeding, blistering, and low diet. At the end of two months he returned to his work as a pavior, but was obliged to desist at the end of a week, owing to general weakness and tingling sensations in his fingers and toes. During the subsequent month the patient only complained of general weakness. The sensory disorders appear to have been in abeyance until they reappeared with greater intensity a few days before the onset of the paralysis. Two suppositions may be made with regard to the development of the symptoms in this case. The first is that the tingling sensations complained of during the week whilst the patient was at work were caused by cold acting directly upon enfeebled and imperfectly nourished peripheral nerves, and that the actual neuritis did not begin until a few days before the appearance of decided paralysis. The second supposition is that the neuritis began during the week in which the patient was at work, and that on desisting there was a partial arrest of the disease, to be followed by a final and fatal recrudescence of it a month later. But there is nothing in either of these suppositions to contradict the view that the case was an example of peripheral neuritis resulting from the poison of diphtheria or some other allied animal poison. Three (36, 73, and 77) out of the nine cases had resulted most probably from alcoholic excess; and in these the premonitory stage lasted three weeks, several months, and two months respectively. It is, however, very usual to meet with a protracted first stage in alcoholic poisoning, and such symptoms as numbness, tingling, and coldness of the hands and feet, and great fatigue on exertion may come and go for years before the accompanying weakness attains to such a degree that it is recognised as a paralysis. In one case (74) the paralysis was the result of syphilis, and the premonitory stage lasted for the unusually long period of four weeks, but a case of multiple neuritis in a syphilitic subject was recently under my observation, in which the onset of paralysis was preceded for some weeks by numbness and tingling of the ulnar fingers and inner borders of the hands, thus showing that the above case is in no way exceptional. In one case (30) the paralysis was caused by a single exposure to the fumes of carbon monoxide, and yet the premonitory symptom of pain along the sciatic nerve lasted 17 days before the onset of paralysis, or at least before it was recognised; but when we come to consider more closely the action of this gas in causing peripheral neuritis, it will be seen that this case also is not exceptional. The remaining three (27, 38, and 64) of the nine cases resulted from unknown causes, and the premonitory stage lasted three or four months, one month, and two months respectively. In the first two of these cases the symptoms, taken as a whole, rather point to alcoholic excess as being the cause of the disease, and if that be true, there would be nothing unusual in their having a protracted first stage. In the third case the premonitory, or at any rate the antecedent, symptoms consisted of an exhausting diarrhœa and weakness, but there are no grounds for believing that the diarrhœa and its attendant weakness formed part of a commencing nervous disorder in the same sense as do numbness and tingling of the extremities. It is, however, possible that both of these symptoms as well as the subsequent paralysis had resulted from prolonged exposure to a poison like alcohol. But whatever view may be taken of the nature of this case there is nothing exceptional or unusual in the presence in it of a prolonged condition of disease antecedent to the onset of the paralysis, and nothing to contradict the opinion that the paralysis was itself the immediate result of an acute multiple neuritis.

(2) *The Stage of Paralysis.*—The sensory symptoms which accompany the motor paralysis being a mere continuation of the sensory disorders of the premonitory stage we shall describe them first. Out of the 90 cases here collected sensory disorders are not mentioned in fourteen cases, and in five cases (34, 39, 65, 72, 86) it is distinctly stated that sensibility was intact, or that there were no marked sensory disorders. In a sixth case (78) it is stated that sensory symptoms were absent, but it is one of the three reservéd for special consideration. Of the fourteen cases in which sensory disorders are not mentioned, nine belong to Landry, and he simply refers to the cases without describing them in detail, and in four more of these cases (42, 49, 56, and 92) the clinical record is brief and imperfect, so that there are no grounds for believing that disorders of sensation were absent in any of these cases, although they are not mentioned. The two remaining out of the fourteen are, one (60) carefully recorded by Westphal, which is of doubtful origin, and another (89) reported by Curschmann, which began

in the course of typhoid fever, and which proved fatal seven days after the first rigor, so that under these circumstances the patient would scarcely be able to describe subjective disorders of sensibility like numbness and tingling. Of the five cases in which it is distinctly mentioned that sensory symptoms were absent the first occurred in a child of eight years of age, most probably after a latent attack of diphtheria, which proved fatal in a few days; the second began after an attack of measles, and terminated fatally in eleven days; the third arose in a syphilitic subject, and proved fatal in nine days; the fourth began during convalescence from typhoid fever, and death occurred on the sixth day; while the fifth was accompanied by fever at its onset, the paralysis reached its maximum intensity in seven days, and remained more or less stationary for the next four weeks, when the patient died from an attack of croupous pneumonia. All of these cases then belong to the group of post-febrile paralysis, and it can hardly be doubted that they own a common pathology with diphtheritic paralysis. The general conclusion we come to then is, that in 71 out of the 90 cases here collected sensory disorders were present; that in 14 cases they are not mentioned, but inasmuch as the clinical record of these cases is imperfect in other ways there are no grounds for believing that they were absent; and that in 5 cases only is it distinctly stated that there were no sensory disorders, these last cases having the common characteristics that they arose suddenly as a sequel to an acute fever, and that the course of the disease was rapid in all and fatal in four of the cases.

The description of these sensory disorders need not detain us long. They consist of the formication, painful creeping, numbness, tingling, feelings of pressure in the fingers, and the other paræsthesiæ in the extremities already described as occurring in the premonitory stage. Pain is a frequent symptom; it is often situated in the extremities, and is then described simply as an aching of the limbs, or more usually as a darting or lancinating pain that shoots along the course of the principal nerve-trunks, which are found tender to pressure. The pain is at other times described as a severe dragging in the loins, as a painful band passing round the chest, or as a painful sensation of constriction passing round the œsophagus. In a few cases mention is made of the presence of hyperæsthesia, and in all of them the muscles were tender to pressure, and it is not improbable, therefore, that the excess of feeling was really seated in the muscles, and not in the skin. In one or two cases pain is mentioned as occurring only on exertion, and in them it probably results from excess of the muscular sensibility; in one of the cases it is accompanied by great restlessness. Pain along or in the neighbourhood of the vertebral column is mentioned in three cases, and heaviness of the vertebral column in one. Heaviness of the vertebral column is a symptom of muscular weakness, while pain in its neighbourhood is most probably due to tenderness of the erector muscles of the spine, for in one case (33) it is distinctly stated that the pain was elicited by "pressure in the *region* of the vertebræ." Coldness of the extremities is a symptom occasionally mentioned, and in one or two cases the hands, or the hands and feet, are said to have been deadly cold, and covered with sweat. Varying degrees of diminution of cutaneous sensibility are frequently present. The diminution is sometimes described simply as a blunting of the cutaneous sensibility, or of the tactile sense; or as a diminution of the cutaneous sensibility, or of the sensibility to pain and touch. In one case (70), Kahler and Pick found an inability to distinguish small differences of temperature; and in another (71), delayed sensibility to pain and a separation of painful and tactile impressions are mentioned, and it is very probable that a careful examination would elicit the presence of these symptoms in most cases. The muscular sense is said to have been lost in a few cases, and analgesia was noted in two (26 and 28), while incomplete anæsthesia of the extremities is mentioned in one case (28), and complete anæsthesia of them in about 10 per cent of all the cases. Visceral pains are not often mentioned, but one patient complained of pain in the epigastrium, and another suffered from cardialgia. The special senses also are only very rarely affected, but in one case the sense of smell is said to have been diminished; in a second, *tinnitus aurium* is mentioned; in a third, amblyopia; and in a fourth, hemianopsia. It is difficult to know what significance to attach to these symptoms, but from the long intemperate habits of the patient (77) affected by hemianopsia, it is very possible that the ascending paralysis was accompanied by thrombosis of a branch of the occipital artery, and consequent softening in the occipital lobe.

Paralysis is generally announced by great weakness of the lower extremities, which soon increases to such a degree that the patient is unable to walk or stand without support. For a short time longer the patient may be able to execute, when lying down, the individual movements of the legs, but this power is soon lost; and in a few days, or even a few hours, every trace of motor power may become abolished in the lower extremities. The legs now lie extended on the bed, while the anterior parts of the feet and toes assume the dropped positions imposed upon them by the action of gravity and by the pressure of the bedclothes; the limbs lie flaccid and powerless, there is no resistance to passive movements, and there is complete absence of muscular tension and contractures.

During the progress of the paralysis in the lower extremities, the upper extremities are attacked. Delicate actions, like those required in writing, become first affected; the gross grasp then becomes feeble, and soon lost; the hands drop at the wrists; and the movements at the elbow and shoulder are successively attacked, and soon rendered impossible.

The muscles of the trunk are now invaded, and the power of sitting up in bed is soon lost; while weakness of the abdominal muscles renders feeble and ineffective the acts of coughing, sneezing, and defæcation; and even the power of rotating the head or raising it from the pillow may be lost from paralysis of the muscles of the neck.

In 41 out of the 90 cases, the muscles supplied by the cranial motor nerves were implicated. The symptoms caused by paralysis of the muscles of the head may be grouped together under the name of bulbar symptoms, if it be understood that this term is used merely for descriptive purposes, and that no theory with regard to the localisation of the lesion is implied. Of the bulbar symptoms, difficulty in swallowing is the most frequently observed, being present in nearly half of the 41 cases in which the muscles supplied by the cranial nerves were implicated. It is generally described as a difficulty or as a slight difficulty in swallowing, but in five cases the inability to swallow was complete. Difficulty in swallowing may depend upon various conditions. The first act of deglutition is rendered difficult by paralysis of the tongue; a second difficulty is caused by the regurgitation of fluids which occurs in paralysis of the soft palate; a third by the passage of fluids and particles of food into the glottis when the epiglottis is paralysed or even becomes anæsthetic; but when mention is made of complete inability to swallow, it may be assumed that the pharynx and œsophagus were paralysed. In one case only (24) is it stated that the pharynx and œsophagus, and in another (49) that the epiglottis and œsophagus, were paralysed. Paralysis of the soft palate is indicated in five cases by the presence of such symptoms as nasal speech and regurgitation of fluids through the nose. In one case (22) the uvula was distorted to the right; in a second (65) it was directed to the left, and the reflex irritability of the soft palate was sluggish; while in a third (6) there was complete immobility of the soft palate. Disorder of articulation is mentioned in 12 cases. It is described in some cases as a difficulty of articulation, and in others as an embarrassment of speech, or, it is said that the speech was indistinct or like that of a drunken person. Disorders of articulation, as apart from defects in vocalisation, may be caused by paralysis of the tongue, lips, or lower jaw. Double facial paralysis was present in five, and paralysis of the tongue, as evinced by inability to protrude it beyond the lips, in three cases. Mastication is said to have been feeble or difficult in three cases, while the masseters along with the orbicularis oris, were paralysed in one (85), and the lower jaw dropped in another case (83). Paralysis of the muscles of mastication, and of the face and tongue, are variously combined in different cases, so that it would be difficult to say how much of the disorder of articulation present in any case is to be attributed to the one and how much to the other, and to attempt the analysis would be a profitless task. In a case reported by Westphal (60), the patient was attacked by sudden speechlessness and paralysis of the right side of the face; but although this mode of onset looks like a cerebral attack, yet the subsequent implication of the muscles of both sides of the face, and of those of the tongue and pharynx, and the fact that no gross lesion was found in the brain on post-mortem examination, seem to indicate that the lesion was situate in the bulbar centres or nerves rather than in the hemisphere of the brain. In two cases (60 and 72) the patient could not open his mouth completely. It is difficult to know what importance to attach to this symptom, as it might have depended upon paralysis of the depressors of the lower jaw, spasm of the masseters, or inflammatory thickening of the temporo-maxillary articulation, but it would be useless to discuss the question at length here.

In addition to the nasal quality imposed upon the voice by paralysis of the soft palate, vocal disorders were present in six cases, which must with probability be attributed to interference with the action of the laryngeal muscles. We say with probability because an indistinct or feeble voice, which are the descriptive terms most usually employed, may have resulted from commencing paralysis of the muscles of respiration. In one case (58), however, the voice is said to have been hoarse, and in another (43) there was complete aphonia, and it is almost certain that these symptoms were caused by a partial or complete paralysis of the adductors of the vocal cords. Paralysis of the ocular muscles was present in five cases. The paralysis is indicated in two cases, by the subjective symptom of diplopia, in a third by external strabismus, in a fourth by double ptosis, and in a fifth by ptosis of the right eye, and double strabismus. Inequality of the pupils was present in two cases.

If the disease advances further the muscles of respiration are attacked; the breathing becomes embarrassed; the epigastrium falls in instead of being curved outwards during inspiration, from paralysis of the diaphragm; the movements of lateral expansion of the chest soon fail, and the breathing becomes of the upper costal type, being carried on wholly by the extrinsic muscles of respiration; but after a short struggle these soon cease to act, and the patient dies from asphyxia.

Paralysis of the respiratory muscles occurred in every one of 53 cases which terminated fatally, although information with regard to the state of the breathing during life is only given in 33 of these cases, and respiratory difficulties are mentioned as having been present in 11 out of 37 cases which recovered, either partially or wholly. It is sometimes definitely stated that certain of the muscles of respiration were para lysed, while at other times it is said that the breathing was embarrassed, or that the patient suffered from dyspnœa, and, in occasional cases, from violent dyspnœa or orthopnœa. In one case (71) the respirations are said to have numbered as high as 50 in the minute before death, but as the evidences of purulent bronchitis and œdema of the lungs were found *post mortem*, it is likely that this high degree of acceleration of the breathing was due to the complication of bronchitis. In contrast with the above is another case (61) in which the respirations only numbered 20 in the minute a short time before death by respiratory paralysis. We believe that the mean of these two extreme cases will represent fairly accurately the degree of acceleration to be usually found in the breathing some hours before the fatal termination. In an acute attack of alcoholic paralysis which we had the opportunity of observing a few days ago, the diaphragm was all but paralysed; the power of expansion of the chest was nearly lost, the difference in the circumference of the chest, between the maximum of inspiration and expiration, being only a quarter of an inch; the breathing was shallow, and of the upper costal type; and the respirations numbered 33 in the minute. There was, however, none of the violent respiratory struggle which is observed in cases of spasmodic and cardiac asthma, bronchitis, and other diseases of the lungs and air passages. The patient lay flat on her back; the countenance, if somewhat dusky, was calm and tranquil, and although the breathing was accelerated, and the epigastrium had fallen in and was only slightly protruded during inspiration, while the action of the upper part of the chest was somewhat heaving, yet the respiratory disorder could have been very readily overlooked by a careless observer, and it is only one specially instructed in such cases that would have been likely to have foreseen that the patient was within a few hours of death. The condition of the breathing in this case may, we believe, be taken as a fair type of what occurs in most other fatal cases. In two cases (60 and 64) the presence of Cheyne-Stokes' respiration is mentioned as a terminal phenomenon.

So far we have only attended to the symptoms which arise from the invasion by paralysis of successive and widely diffused groups of muscles, but let us now attend to the characteristics of the paralysis, as it affects individual parts.

Disorders of reflex action always accompany this form of paralysis. With regard to the cutaneous reflexes, information is given only in 24 out of the 90 cases here collected. In two of these (82 and 85) it is stated that the reflex of the sole, and in one case (87), that the cutaneous reflexes generally were retained. In four out of the remaining 21 cases, the reflex of the sole is stated to have been lost, and in one case the reflexes of the sole and cremaster were absent, while in 2 cases it is stated generally that the reflexes were diminished, and in 14 that they were lost.

No mention is made of the condition of the knee-jerk before the publication of Westphal's (60) papers in 1876, inasmuch as before that date the value of this reaction as a sign of disease had not been ascertained. Since that time mention is made of the state of this reaction in 16 cases, and in all of these the knee-jerks were completely lost, but in one case (88) they soon reappeared.

There is no statement which is so persistently made in books as that the electrical reactions remain normal in this disease, but an analysis of our cases will show that it rests upon very slender evidence. The electrical reactions were noted in 32 only of our 90 cases. In 12 of these 32 cases there is a general statement as to the state of the electrical reactions without specifying the kind of current employed; in 11 cases the nature of the reaction to the faradic current is alone mentioned; and in other 9 cases the reactions obtained from both currents are reported. Of the 12 cases in which the electrical reactions are alone mentioned they are said to have been lost in three (64, 84 and 92), diminished in one (85), and normal in 8 cases. But a careful examination of the reports of the cases in which it is said that the electrical reactions were normal leads one to the conviction that in most of them, at least, the faradic current was alone used as a test, so that all these cases prove beyond doubt is that in them the faradic irritability of the affected nerves and muscles was not markedly diminished. Of the 11 cases in which the faradic irritability of the nerves and muscles was alone noted it is said to have been normal in 8, diminished in 1, and lost in 2 cases. The 9 cases in which both faradic and galvanic reactions were both noted deserve to be considered with some degree of care. In one of Bernhardt's cases (50) the electrical reactions were normal for eight days, but during the further progress of the case the faradic contractility became progressively diminished, and ultimately completely lost in the peroneal muscles and extensors of the foot. In the muscles which failed to respond to the faradic current the galvanic reactions passed through different phases of activity, in which a brief period of diminution was followed by successive stages of great increase, gradual diminution, disappearance, and reappearance with gradual increase, to end, so far as the observations go, in a final stage of increased activity. There is, however, no mention made of the degree of sensitiveness manifested by the muscles to the respective poles on closure and opening, but enough is said to prove conclusively that in this case some of the nerves and muscles manifested the complete reaction of degeneration. A confirmation of this opinion is to be found in another way. The mechanical irritability of the muscles most affected was increased in degree, and a blow over one of them was followed by a contraction which reached its maximum intensity slowly and then gradually disappeared, this being the kind of contraction to be obtained in muscles which have undergone a considerable degree of degeneration. In the above patient the stage of paralysis is said to have lasted only seven days, but convalescence was slow, and at the end of twelve months the patient had double ankle drop and the movements of the upper extremities were feeble. The symptoms bear the stamp of alcoholic poisoning and the electrical reactions obtained correspond with what is usually met with in severe cases of this form of paralysis Bernhardt's next case (51) was also an example of alcoholic paralysis, and the stage of paralysis lasted twelve days, but the patient made a fair recovery in two months, so that the degree of paralysis was not very profound. In this case the faradic irritability is alone mentioned, and it is included amongst the cases in which the reactions to this current were normal, although the author admits that they were probably diminished in the interossei muscles. In another of Bernhardt's cases (52) the nerves and muscles most affected gave diminished reactions to both the faradic and galvanic currents, and in this case the presence of the partial reaction of degeneration may safely be assumed. The case was probably also one of alcoholic paralysis; the stage of paralysis is said to have lasted only two days; but convalescence was slow, and only very imperfect recovery took place in two months, while the muscles of the hands had undergone atrophy. These three cases of Bernhardt's are here mentioned in detail, because we imagine that they may, so far as the electrical reactions are concerned, be taken as types of all the other cases of this disease in which recovery takes place. The first of these cases was an example of a profound degree of paralysis with a protracted stage of convalescence, and in it the complete reaction of degeneration was noted; the third was an example of a moderate degree of paralysis, with a corresponding duration of the stage of convalescence, and in it the partial reaction of degeneration was observed, while the second was an example of a minor degree of paralysis, followed by a short stage of convalescence, and in it the reactions were found to have been normal, or nearly so.

Let us now turn to Westphal's cases, and these also will be here examined in detail, partly because of their intrinsic importance, and partly because he was the first author to lay down the dictum that in Landry's paralysis the electrical reactions are normal. In his first case (58) it is stated in the tables that the electrical reactions were normal, but on referring to the original report of the case we find it stated that both the faradic and galvanic reactions were repeatedly tested in the course of the disease, by Remak, and found normal. The competence of the observer in this case is beyond doubt, and the observations themselves acquire additional significance from the fact that although the disease proved fatal in the usual way by asphyxia, yet the paralytic stage lasted for the comparatively long period of 28 days. The patient had an attack of hæmoptysis three years before the onset of the disease, and at the autopsy grey tubercle was found scattered throughout both lungs; and notwithstanding that the electrical reactions were found normal, we see no reason to doubt that it was an example of the peripheral neuritis which is now known to occasionally accompany tuberculosis. In Westphal's second case (59), the faradic irritability was repeatedly tested, but the galvanic irritability was only once tried at the beginning of the disease, and at a time, therefore, in which a degenerative reaction could not be expected to be present. This case is then included amongst those in which the faradic irritability was alone observed, and Westphal's remaining two cases (60 and 61) belong to the same category.

In the case (66) reported by Jaffé, the electrical reactions, examined by Erb, were at first normal, but as the case progressed the faradic contractility of some of the nerves and muscles became gradually diminished and finally lost. The galvanic irritability of the muscles was also diminished, but there was no reversal of the normal formula of contraction in the muscles. The most remarkable feature about this case, which was probably an alcoholic paralysis in a syphilitic subject, is that the faradic irritability was lost in a patient who died at the end of ten days from the onset of the first premonitory symptoms. In Kahler and Pick's first case (70) the faradic reactions were normal, but there was a slight diminution of the galvanic reactions in some of the muscles of the lower extremities, but without change of formula. The case was probably of diphtheritic origin, and the paralytic stage only lasted eleven days. In their second case (71) the faradic and galvanic reactions were normal. This case was one of alcoholic paralysis, which could only have been of a moderate degree of intensity, as the patient recovered completely in three months. In Schulz and Schultze's case (74) the faradic contractility was lost, and the galvanic reactions gave the degenerative formula. The lesion occurred in a syphilitic subject; the stage of paralysis lasted 23 days, and the gradual recovery that was taking place was interrupted by an attack of bronchitis, which proved fatal. In Myrtle's case (77) the galvanic irritability was lost. The case was one of alcoholic poisoning, and the paralytic stage lasted forty days. The last case of this group is the one reported by Dr. Dixon Mann (91), and in it the faradic and galvanic reactions were found normal. The cause of the disease was doubtful, but it pursued a rapidly fatal course, the paralytic stage only lasting five days. To sum up the result of our analysis of these nine cases, the faradic and galvanic irritability manifested, in two cases (50 and 77) the complete reaction of degeneration, and in three (52, 66, and 74) the partial reaction of degeneration, while in four cases (58, 70, 71, 81) the reactions to both currents were normal. These last cases show that the electrical reactions may remain normal when, as in two of the cases (70 and 81), the disease runs an acute and fatal course, and when, as in another case (71), the paralysis is not so profound in degree, but that the patient makes a moderately quick recovery; but there is nothing anomalous in these cases, on the theory that the disease is a peripheral neuritis. The fourth case (58) shows that a paralysis may prove fatal by implication of the respiratory muscles, when an electrical examination has proved that the nerves and muscles of the extremities are not profoundly affected by the disease, although there is a widely diffused and complete paralysis for the time. And here again there is surely nothing very anomalous or surprising, and nothing which would lead us to separate this case in its pathology from cases of peripheral neuritis, with which it is clinically allied. Putting together the 12 cases in which information is obtained about the electrical reactions generally, and the 11 in which the faradic reactions are alone mentioned, the irritability is said to be lost in five, diminished in two, and normal in sixteen cases. It may be assumed that a complete reaction of degeneration was present in the five cases in which the reactions were lost, and a partial reaction of degeneration in the two in which it was diminished. In the sixteen cases in which the electrical irritability was said to have been normal, the faradic current was alone used as a test in by far the majority of the cases; but retained faradic irritability is quite compatible with the presence of that inversion of the normal formula of galvanic contractility which constitutes the reaction of degeneration in its partial form; and it is this partial form of degenerative reaction which is probably most frequently met with in cases of peripheral neuritis. A survey, therefore, of all the cases in which information is obtained with regard to the electrical reactions in Landry's paralysis proves that the statement made in books as to their being always normal is unfounded, and a further study of peripheral neuritis will convince us that the absence of any alteration in these reactions in a few cases is not so anomalous a condition as has hitherto been supposed.

In acute ascending paralysis the general health is, as a rule, good, and it is quite exceptional for the disease to be ushered in or accompanied by fever, but it is very probable that a slight rise of temperature occurs in most fatal cases during the stage of respiratory paralysis. The slight elevation of temperature which occurred in Cases 64 and 73, may admit of this explanation. In the case reported by Gombault (47), fever, for which it is difficult to find an explanation, is said to have been present during the first week, and in the case reported by Calestri (54), fever and diarrhœa came on in the course of the disease, but as the patient died 18 days after the paralysis ceased to progress, from an attack of acute gastro-enteritis, and as he had previously suffered from an attack of acute rheumatism, and evidences of endocarditis, pericarditis, and hypertrophy of the heart were found after death, the febrile symptoms and diarrhœa must be regarded as being due to some complication. In one of Westphal's cases (58) the patient had a rigor in the course of the paralysis and the temperature rose to 40·4°C. and continued afterwards to be of remittent type, but these symptoms found an ample explanation *post mortem* in the presence of grey tubercle being scattered throughout both lungs. In Curschmann's case (89) of paralysis occurring in the course of typhoid there was high fever.

The functions of the bladder were disordered in 14 cases. In Landry's case the bladder is said to have been distended, and in four cases retention of urine was present. In three cases incontinence of urine is mentioned; in two, the bladder or its sphincter is said to have been paralysed; and in four cases the bladder is said to have been weak, feeble, or slightly affected, or the urine to have been passed with difficulty. It is most likely that in the majority of the cases the bladder was only secondarily affected by the inability of the patient to aid the expulsive efforts, owing to paralysis of the abdominal muscles and the diaphragm. And even when incontinence is said to have been present, it is possibly only the incontinence of an over-distended bladder. In one case (88) in which the stage of paralysis lasted twenty-nine days, and of convalescence five weeks, the patient is said to have been impotent. Mention is made of disorders of the functions of the bowels in seventeen cases; in nine the bowels were obstinately constipated; in two diarrhœa was present; in two there was incontinence of fæces; in two the sphincter is said to have been paralysed; and in the remaining two cases the sphincter is said to have been slightly affected, or weak. It is very probable that at least the constipation is caused, not so much by paralysis of the muscular coat of the bowels, as by the inability of the patient to strain, owing to advancing paralysis of the abdominal muscles and diaphragm; but incontinence of fæces is most probably due to direct implication of the sphincter in the paralysis.

The condition of the pulse is mentioned in eleven cases, and in frequency it will be seen to have varied from 62, the lowest, to 160 as the highest number of beats in the minute. In one of the cases reported by Kahler and Pick (71), for example, the pulse beat at the rate of 80 in the minute during the first days of the paralysis, but rose to 160 before death, and it is very probable that this case may, so far as the rate of the pulse is concerned, be taken as a type of most fatal cases.

Trophic disorders do not form a prominent feature of the disease. In one case (61) a slight bed sore had formed on the sacrum, and in another (53) the joints of the left hand were swollen. In a third case (41) numerous purpuric spots appeared on the toes, but it is probable that both the purpura and the paralysis were the result of the infection of the patient by syphilis five years before the attack. The occurrence of trophic changes in the muscles will be discussed when we come to describe the stage of convalescence. The only vaso-motor disorder mentioned, besides the coldness of the extremities already described, occurred in one case (77), and consisted of patches of vascular congestion, like the tâches cérébrales of epileptic seizures.

Psychical disorders are almost always absent, the mind being in by far the majority of cases quite clear and calm to the last. In one case (16), however, the patient is said to have been apathetic; in a second (35) there was mental confusion, and in a third (45) sleeplessness. In one case (73) the patient was delirious; and in another (64) delirium passed into sopor; while in a case of alcoholic paralysis (77) the patient had epileptiform seizures, and passed into a rambling and semi-conscious condition.

The complications which occur in the course of the disease are not numerous. In one case (22) there was dulness of both apices of the lungs and a sudden discharge of stinking pus; in a second (3) there was loss of appetite and diarrhœa; in a third (39) an attack of pneumonia developed near the fatal termination; in a fourth (59) rhonchi were heard over both lungs; in a fifth (54) hypertrophy of the heart was noted in association with mitral insufficiency, enlarged spleen, diarrhœa, and fever, and death resulted from acute gastro-enteritis; and in a sixth case (73) the urine was albuminous.

(3) *The Stage of Convalescence.*—This stage does not demand a lengthened description. The paralysis may cease to advance at any stage of its progress, and even after the most threatening symptoms of respiratory paralysis have become established, and then after a stationary period of from a few hours to two or three weeks, improvement sets in, and motor power is gradually regained, the muscles last attacked being the first to recover, so that recovery takes place in the reverse order to that in which the muscles were implicated. The muscles of respiration, and of the head and face, first recover motor power, then in succession the muscles of the abdomen, the erectors of the spine, the larger muscles which pass from the trunk to the limbs, the muscles of the proximal segments of the limbs, and finally those of the distal segments. A stage of convalescence was established in 36 out of the 90 cases here collected. In one case (86), convalescence was interrupted at the end of four weeks by an attack of pneumonia, which proved fatal. In another case (48)

death was caused seven years after the occurrence of the paralysis from cancer of the abdomen, but convalescence can hardly be said to have been interrupted by an inter-current disease in this case. The duration of the stage of convalescence has varied from a few days to one or two years, and it is worthy of note, that neither the duration of the convalescence, nor the completeness of the recovery bear any relation to the apparent danger of the symptoms during the paralytic stage. In Case 50, for example, the stage of advancing paralysis is said to have lasted only 7 days, and the muscles of the extremities appear alone to have lost their motor power, yet the patient could not stand without support at the end of 12 months. The case (87) reported by Sorgenfrey forms a strong contrast to the above. In it the paralysis lasted 4 days only, but not only were all the limbs paralysed, but there was loss of voice, difficulty of deglutition and of articulation and dyspnœa, and yet complete recovery took place in 7 days. If it be said that in Sorgenfrey's case the disease must have been, from its transitory character, functional and of the nature of hysteria, we may take the case (90) reported by Möbius, and which is an undoubted example of Landry's paralysis. The stage of paralysis lasted for the comparatively long period of 24 days, and in addition to the muscles of the extremities those of the neck were paralysed; the voice was weak, and even the diaphragm was completely paralysed, and yet the patient made a complete recovery in two or three months. The truth is that the length of the stage of convalesence must depend upon the degree of morbid change which takes place in the affected nerves and muscles, while the danger to life depends upon the wide diffusion of the paralysis and upon the implication of muscles necessary to life, like those of respiration.

We have already seen that it is persistently stated in books that the electrical reactions are normal in this disease, and another statement as frequently made and equally unfounded with it, is that the muscles do not undergo active atrophy. In the stage of paralysis a large number of the muscles of the body are so uniformly attacked, and especially those of the extremities where wasting of them is best observed, that it is difficult to distinguish active atrophy from the general wasting which accompanies all acute diseases. The limbs are so flaccid that they assume the positions imposed upon them by the action of gravity, checked only by the rigidity of the bones and the action of the ligaments, and thus the distortions that occur when a few only of the muscles are paralysed, are absent. And when the stage of convalescence is comparatively short the recovery of motor power proceeds more or less uniformly, so that the distortions of the limbs which occur in chronic muscular atrophies do not form a prominent feature of the case. But that these distortions are present in the cases in which the stage of convalescence is protracted there can be no doubt, and we believe they will be found in almost all cases if carefully looked for. Be this as it may, we find in the case recorded by Cumming (40), in which the stage of convalescence was very prolonged, that the *main en griffe* persisted at the end of two years. In the cases reported by Gombault (47 and 48), again the stage of convalescence was prolonged, and in the first there was persistent atrophy of some of the muscles, while in the second atrophy of the muscles of the hands and feet appeared at the end of three-and-a-half months, and the consequent deformities persisted. In one of the cases (50) reported by Bernhardt the patient had double ankle-drop at the end of twelve months, while in a second (52), atrophy of the muscles of the hands was noted. At the onset of our analysis, we reserved three cases out of the 93 here collected for special consideration, and two of them (78 and 79), which are reported by Schultze, we now propose to consider. In these cases the paralysis pursued an ascending course, and terminated fatally by respiratory paralysis at the end of six months in the one and of seven months in the other. The chief characteristics, however, of these cases to the mind of the reporter are that some of the affected muscles had undergone atrophy, and that the nerves and muscles manifested the reaction of degeneration on electrical examination. He believes that the presence of these symptoms justifies him in separating his cases from Landry's paralysis, and in placing them in a class of their own, which he proposes to name "ascending atrophic paralysis." Now a careful examination of the reports of these cases proves to our mind that they are clear examples of what we now regard as multiple neuritis, the one arising in a syphilitic subject and the other from unknown causes; and we believe that such cases are connected by every variety and degree of intermediate form with the acute cases which are regarded as forming the type of acute ascending paralysis, and in which the muscles do not undergo recognisable atrophy, and the nerves and muscles do not give abnormal electrical reactions.

Course, Duration, and Terminations.—The statement usually made with regard to the advance of the paralysis in this disease is that it pursues an *ascending* course, this being qualified by the admission that in occasional cases the course is a *descending* one. The assertion, respecting the *ascending* course of the disease, is either true or not true, according to the sense in which the term is employed. The word "ascending" was used by Landry simply for the purposes of clinical description, and what he implied by it was that the paralysis first attacked the muscles of the peripheral segments of the limbs—the small muscles of the hands and feet—and then gradually ascended to the muscles of the proximal segments, and finally to those of the trunk, and in this sense he correctly used the word "centripetal" as a synonym for "ascending."

The muscles, according to Landry,* are attacked in the following order: (1) the muscles which move the toes and feet, then the posterior muscles of the thigh and pelvis, and lastly, the anterior and internal muscles of the thigh; (2) the muscles moving the fingers, the hand, the arm upon the scapula, and lastly, the forearm upon the arm; (3) the muscles of the trunk; and (4) the muscles of respiration, and then those of the tongue, pharynx and œsophagus. Now although the order in which the muscles are invaded, may vary considerably in individual cases, even amongst those which are supposed to pursue a typically regular course, yet we believe that the order assigned by Landry is as correct as any single description can be expected to be, and if so, the term ascending, in the sense of centripetal, is not an inapt one. This term has, however, acquired a secondary meaning for which there is no warrant whatever, and which makes it convey a quite erroneous idea when applied to this disease. The term ascending, which was used by Landry as a clinical description, is transformed so as to prop up a theory as to the anatomical seat of the disease. By the use of this word it is tacitly implied, although perhaps not often openly expressed, that the muscles are invaded in the order in which they are innervated from the spinal cord, the disease beginning at its lower part, and gradually ascending until the medulla is reached. This general statement is supplemented by the subordinate assertion that the paralysis sometimes begins in the upper extremities, and then pursues a descending course, the implication being that the muscles are invaded in the order in which they are innervated from the cord passing from above downwards. Now it is true that this anatomical meaning of the words ascending and descending may be quite correctly applied to certain forms of central myelitis, but it is not true that they can be applied, as thus defined, to describe correctly the course of the symptoms in Landry's paralysis.

In 47 out of the 90 cases here collected, no mention is made of paralysis of the muscles supplied by the bulbar nerves. In 15 out of these 47 cases it is merely mentioned generally that the paralysis pursued an ascending course, or that the extremities were feeble, while in 20 cases it is stated either that the lower extremities were the first to be paralysed, or that they were more profoundly affected than the upper extremities. In 10 of these cases the lower and upper extremities appear to have been attacked simultaneously. In one case (36) the lower extremities were alone affected; and in another (40) the upper extremities were attacked before the lower. In 43 out of the 90 cases, some at least of the muscles supplied by the bulbar nerves were paralysed. In 30 of the 43 cases the lower extremities were the first to be implicated; in 26 of these the upper extremities were attacked next, and the bulbar nerves last; in one case (58) the upper extremities and bulbar nerves were simultaneously attacked; and in the three remaining cases (61, 72, and 85) the bulbar nerves were implicated before the upper extremities. In seven of these 43 cases the lower and upper extremities were simultaneously attacked, and the bulbar nerves were the last to be invaded. In the remaining 10 cases the bulbar nerves were implicated at the onset of the symptoms; in one (18), the legs were the next to be attacked, and the arms last; in two (29 and 34), the arms were second and the legs last; in one (60), the arms and legs were simultaneously attacked after the bulbar nerves; and in two (39 and 51) the limbs were invaded at the same time as the bulbar nerves. This analysis, imperfect though it be, shows conclusively that the paralysis does not pursue an ascending or a descending course in the sense that the muscles are attacked according to the order in which they are innervated from the spinal cord; that the word ascending is only applicable to the course of the disease in the sense of centripetal assigned to it by Landry; and that the word descending is erroneous as descriptive of the symptoms either in the anatomical sense that the muscles are attacked according to the order in which they are innervated from the spinal cord from above downwards, or when used as the opposite of centripetal to imply that the muscles of the trunk are the first and the peripherally placed muscles the last to be attacked.

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* Landry (D.). *Loc. cit.* (6), p. 487.

With regard to the duration of the disease, the cases in which it proved fatal must be considered apart from those which recovered. Out of the 90 cases here collected, 52 died during the stage of paralysis, and 38 made a partial or complete recovery, and of the last group three died from an intercurrent disease during the stage of recovery, and one many years later. In 28 out of the 52 fatal cases bulbar symptoms were present. In 16 of these the bulbar nerves were attacked last, and the stage of paralysis is said to have lasted from two days to several months, the average being 40 days; in 8, they were the second to be attacked, and the paralysis lasted from three to 28 days, the average duration being 14 days; while, in 4 cases, they were the first to be attacked, the stage of paralysis lasting from three to 14 days, the average being 8 days. In 24 out of the 52 fatal cases no bulbar symptoms are mentioned, and in these the stage of paralysis is said to have lasted from three days to several months, the average being 13 days. Of the 38 cases which recovered, 12 made a partial and 26 a complete recovery. Of the 12 cases which made a partial recovery, the stage of paralysis lasted from two days to two months, and the average duration was about 16 days. With regard to the duration of the stage of convalescence in the cases which made a partial recovery, the patients remained under observation for a variable period of from a few weeks to a few years. In three of these cases death was caused by an intercurrent disease during the stage of recovery; in the first case (54) the paralysis ceased to progress at the end of a month, and the gradual recovery which began was interrupted at the end of 18 days by an attack of acute gastritis, which quickly proved fatal; in the second (74), the stage of paralysis lasted 23 days, and the convalescent stage was interrupted at the end of another 21 days by a fatal attack of bronchitis; and in the third (86), the stage of paralysis lasted seven days, and the convalescent stage was interrupted, at the end of four weeks, by a fatal attack of pneumonia. In one (10) of these 12 cases the stage of paralysis is said to have pursued an acute course, but the length of time during which the patient remained under observation is not stated. In two (51 and 52) of the cases the stage of paralysis lasted 12 and 2 days respectively, but the patients remained under observation during the stage of recovery only for a period of two months each, and it is, therefore, probable that these patients had subsequently made a complete recovery. Other cases which may have made a complete recovery are one (36) in which the stage of paralysis had lasted 10 days, and only partial restoration took place in six months; and another (45) in which the stage of paralysis lasted 28 days, and partial restoration was effected in 5 months. Four cases remain out of the 12 in which partial recovery had taken place. In one of them (40) the stage of paralysis lasted 2 or 3 weeks, and at the end of two years the *main en griffe* persisted; in the second (47) the stage of paralysis lasted a month, and at the end of a prolonged period some of the muscles remained persistently atrophied; in the third (48) the stage of paralysis lasted only 5 days, but atrophy of some of the muscles of the extremities, with consequent deformities of the hands, was found 7 years later, when the patient died of abdominal cancer; and in the fourth (50) the stage of paralysis lasted only 7 days, yet at the end of a year the patient could not stand without support, and the hands were deformed. In these four cases, then, it is clear that some of the muscles had become permanently atrophied and paralysed, and yet in two of them the stage of paralysis only lasted 5 and 7 days respectively, so that there does not appear to be any direct proportion between the length of the stage of paralysis and of that of convalescence. In the 26 cases which made a complete recovery the stage of paralysis varied from 2 days to six weeks, the average duration being about 13 days. The stage of convalescence lasted from 14 days (11), or a few days (35), to a period of 7 (80) or 9 months (2), but in the great majority of cases complete recovery took place in from 2 to 5 months. It has already been shown that bulbar symptoms were present in 29 out of the 53 fatal cases, and it may now be stated that these symptoms were present in one only (51) of the 12 cases which made a partial, and in 6 of the 25 cases which made a complete recovery. It would appear, then, that bulbar symptoms were noted in about 56 per cent of fatal cases, and in about 19 per cent of those which recovered, thus showing that the presence of these symptoms adds to the gravity of the prognosis, although their appearance is by no means of fatal significance.

With regard to the terminations of the disease, it has already been mentioned that 37 cases recovered either partially or completely, and 53 died in the stage of paralysis. Enough has been said with regard to the terminations in the cases which recovered, and now it only remains for us to consider the mode of death in the fatal cases. In three of these cases the patients were slightly delirious before death, but the mode of dying is not mentioned. In the first case (22) there was a sudden discharge of stinking pus from the lungs a few days before the patient died, and syphilitic cicatrices of the liver were found post mortem, so that the patient may have died from pyæmic infection. In the second (30), the paralysis resulted from exposure to the fumes of carbonic oxide, and it would therefore be complicated by great anæmia from destruction of the hæmoglobin; and in the third (64), the patient was exhausted before the onset of the paralysis by a continuous diarrhœa which recurred before the fatal termination, and was then accompanied by a rise of temperature to 39·3° C., and whatever might be the nature of this diarrhœa it is to this complication most probably that the delirium was due. In one (77) of the fatal cases the patient died comatose. The patient had been addicted for many years to excessive drinking, and it is probable that at the end the paralysis was accompanied by a cerebral complication.

In another (72) of the fatal cases the patient is said to have died from œdema of the lungs and paralysis of the vagus, but whether the mode of dying was by respiratory paralysis or cardiac failure is not stated. In seven cases it is simply stated that the disease proved fatal, but the mode of death is not mentioned. In the remaining 41 out of the 53 fatal cases death was caused by respiratory paralysis; in two of these cases (58 and 61) the breathing before death is said to have been of the upper costal type, so that the extrinsic muscles of respiration were the last to be paralysed; in seven it is distinctly stated that the diaphragm was paralysed before the other muscles of respiration; in two cases (76 and 84) it is said that the breathing before death was carried on entirely, or almost entirely, by the diaphragm, and in the remaining 30 cases the patients are said to have died from suffocation or respiratory paralysis, but the order in which the respiratory muscles were attacked is not mentioned. The above analysis thus renders it clear that the natural termination of this form of paralysis, when fatal, is by asphyxia, and it is probable that, in by far the majority of cases, the diaphragm is the first respiratory muscle to be attacked, although it is likely that the intercostal muscles are at the same time more or less affected, while the extrinsic respiratory muscles hold out to the last.

Morbid Anatomy.—It has already been stated that out of the 90 cases here collected, 52 died and 38 made a partial or complete recovery. A reference to the tables will show that a post-mortem examination was obtained in 38 of the 52 cases that died during the stage of paralysis; also in three cases that died from an intercurrent attack of acute disease during the stage of convalescence, and in one case that died from abdominal cancer seven years subsequent to the paralytic seizure, making in all 42 cases in which an autopsy was obtained. Now it is not necessary to pass all of these cases under careful review. Congestion of the cerebral and spinal membranes is said to have been present in several cases, but the mode of dying by asphyxia would cause considerable congestion of these membranes, and the appearance of distended vessels post mortem affords no evidence that the membranes were the subject of active disease. In cases recorded by Ollivier (1), Brochin (5), Landry (6), Leroy d'Etiolles (17), Pellegrino-Lévi (29), Leudet (31), Oulmont and Hayem (37), Bourdillat (39), Chalvet and Kiener (43), Duchenne (46), Calestri (54), Westphal (58 and 59), Kahler and Pick (70), Finny (76), Ross (82), and Mann (91)—making, in all, 17 cases—the results were either entirely negative, or no evidence of disease beyond a slight degree of congestion of the membranes was discovered. In eight of these cases a careful microscopical examination was made of the nervous centres by skilled observers. So far as the evidence of these cases is concerned, then, it would seem to show that the symptoms of this disease are not caused by a primary lesion of the nerve centres, or, at least, not by any more profound lesion than a functional disturbance of them. In cases recorded by Pellegrino-Lévi (27), Bablon (29), Bernhardt (44), and Westphal (60), in addition to the nerve centres, some of the nerve roots and nerve trunks were examined, and found healthy, and in three of these cases a microscopical examination was made. In one of the cases reported by Walford (3), and the one recorded by Harley and Clarke (38), portions of the spinal cord were found to have undergone softening, but, as has been pointed out by Leyden, it is more likely that the softening had resulted from post-mortem changes than from disease.

In one of the cases reported by Kussmaul (22), syphilitic cicatrices were found in the liver, and the cerebral dura mater was thickened, but the spinal cord was normal, and consequently this case may be added to those in which the results of the autopsy were negative, inasmuch as disease of the cerebral membranes, with secondary implication of the cortex of the brain, would not give rise to this peculiar kind of paralysis with loss of the reflex actions.

Putting together the 17 cases in which the brain and spinal cord were alone examined, the four cases in which the brain, cord, and peripheral nerves were examined, the two cases in which softening of portions of the cord were observed, and the one in which thickening of the cerebral dura mater was noted but the cord was normal, they make 24 cases in which the results of the post mortem may be said to have been negative.

The cases in which positive changes were discovered must now be subjected to careful analysis. In the case (30) of carbonic oxide poisoning, reported by Leudet, the patient suffered during the initial stage from pain and tenderness over the right sciatic nerve, and signs of neuritis of that nerve were discovered at the autopsy, but the brain and spinal cord were found normal. In Gombault's case (48) the patient made a partial recovery, but atrophy of the extensors of the forearms and legs with consequent deformities, persisted for the remainder of life, death being caused seven years after the paralysis by abdominal cancer. On microscopical examination, Gombault found that the ganglion cells of the anterior grey horns were globular in form and contained an excess of pigment. He also found degenerative fibres in the anterior roots, and islets of sclerosis with excess of connective tissue, in the nerves supplying the affected muscles. In one case (55) reported by Leyden, a small focus of disease was found in the right half of the medulla oblongata on a level with the nuclei of origin of the glosso-pharyngeal, the vagus, the spinal accessory, and, in part, the facial nerves, while there was a rich infiltration of cells and granules in the tissue surrounding the central canal of the cord. In a second case (56) the same observer found a cystic softening of a portion of the cervical region of the cord with sclerosis of the tissue surrounding the central canal. In the case (57) recorded by Déjerine and Goetz, the spinal cord was found to be normal, but degenerated fibres with multiplication of nuclei were found in some of the anterior roots. From this case the authors argue that the primary disease began in the ganglion cells of the anterior horns, although the process was not of sufficient intensity, or long enough in duration to have left traces of disease in the cells themselves. In a case (61) of post-diphtheritic paralysis, recorded by Westphal, the spinal cord was found normal, but the anterior crural nerve was found on section to present changes similar to those found by the same observer in the musculo-spiral nerve in a case of lead paralysis. In a very important case (64) reported by van der Velden and Leyden, certain foci of myelitis were found in the spinal cord, and in these the axis cylinders of the nerve fibres were found swollen. Some of the ganglion cells were found to be swollen and pigmented, while others were atrophied, but the cells did not manifest a multiplication of their nuclei. Minor changes were also met with in some of the nerve trunks. In the case (65) reported by Eisenlohr morbid changes were found on a level with the third cervical and upper lumbar nerves, consisting of an accumulation of cells round the central canal and pigmentation of the ganglion cells of the anterior horns. Capillary hæmorrhages were found in the medulla oblongata and pons, and minute foci in the pons in which there were an accumulation of lymphoid cells. In cases recorded by Cornil and Lépine (69), Fox (73), Ross (81), and Immermann (86), minor changes were found in the cord, but the peripheral nerves were either not examined or found healthy. In the case recorded by Kümmell (72) small hæmorrhagic foci were found in the pons, but the cord was normal. In the case recorded by Schulz and Schultze (74), in addition to slight changes met with in the spinal cord, minor changes were observed in the peripheral nerves as well; and in the case recorded by Hoffmann (85), evidences of inflammation of the cord and medulla oblongata and of their membranes were noted, while degenerative changes were likewise observed in the right facial nerve, but the other nerves were normal. It would then appear that definite morbid changes of the spinal centres, nerve roots, or nerve trunks were observed in 15 of the 42 cases in which an autopsy had been obtained. To find a change, however, in the nervous tissues is not enough; in order that the change may be regarded as the essential one, it ought to have been encountered in a considerable number of cases, or, if found in a few only, it ought to be of such a nature and localisation as will explain the most prominent symptoms of the disease. Now, the presence of small hæmorrhages or foci of accumulated leucocytes in the pons and medulla oblongata does not fulfil either of these conditions. These lesions have, in the first place, only been found in a very few cases, although their presence would be by no means difficult to discover; and, in the second place, had they been met with in all cases, the facts that the paralysis generally begins in the lower extremities, and that the cutaneous reflexes and tendon reactions are lost in them at an early stage in the progress of the case, would still be unexplained. Without denying, then, that foci of disease have been observed in the medulla oblongata and pons in a few cases of Landry's paralysis, it may be safely asserted tha' they are only concomitant to some other lesion which is essential to the production of the chief symptoms. The case reported by Kümmell (72), in which a small hæmorrhage was discovered in the restiform body, while the cord is said to have been normal, may be dismissed from our consideration; but the three other cases (55, 65, and 85), in which lesions were formed in the medulla or pons, must still be retained, inasmuch as, in addition to these, other changes were observed in the spinal cord. Of the 14 cases which remain, the spinal cord was found the seat of morbid changes in 11, and normal in three cases. Of the 11 cases in which the spinal cord was found diseased, the lesion in one, reported by Leyden (56), consisted of a cystic softening in the cervical region, with a pericentral sclerosis; and in a second case (55), reported by the same author, it consisted of an infiltration of cells and granules round the central canal. Now, whatever may be the significance of these pericentral changes, it is clear that the cystic softening of the cervical region is liable to all the objections urged against lesions in the medulla oblongata and pons as the anatomical cause of the disease; this kind of softening has only been found in one out of the many cases examined, and were it found in all it would not afford an explanation for the leading symptoms. With regard to the pericentral changes described by Leyden, it will suffice to say that the condition of the tissue surrounding the central canal varies greatly in perfectly normal cords, according to the age of the patient and other circumstances, and that in the absence of any definite changes in the ganglion cells it would be hazardous to accept these changes as a sign of disease. It may also be remarked that in these two cases reported by Leyden the symptoms are not recorded in sufficient detail to enable us to be quite sure that the cases were real examples of the acute ascending paralysis described by Landry, information with regard to the state of the cutaneous reflexes and tendon-reactions being wholly wanting. In the case (85) reported by Hoffmann, the observer states that although he found evidences of a myelo-meningitis in the greater part of the spinal cord, yet the lumbar enlargement was free from disease, and consequently this case may be put on one side, as the localisation of the lesion does not afford an explanation of the absence of the patellar-tendon reactions in this disease. In the eight remaining cases certain changes are described as having been observed in the grey matter of the spinal cord, and more especially in the ganglion cells of the anterior horns. Some of the ganglion cells are said to have been swollen, and to have contained an accumulation of pigment granules, or to have presented vacuolation, but the more experience I have acquired in the examination of sections of healthy and morbid spinal cords, the less importance am I inclined to attach to these changes as signs of disease. All of them can be seen in perfection in the spinal cords in cases of tetanus and hydrophobia, but no one now regards these diseases as being due to a myelitis. Amongst my own preparations I have retained a well-preserved section from the spinal cord of one (81) of the cases recorded by myself, and on examining it to-day I still find that some of the ganglion cells are swollen and pigmented, and that there is a rich infiltration of cells round the central canal, but there are no destructive changes at all comparable to those to be observed in sections of the cord from acute central myelitis, chronic polio-myelitis, and amyotrophic lateral sclerosis in my possession. In my own case, therefore, I abandon the theory of acute central myelitis, or acute anterior poliomyelitis, and I do not see that the post-mortem evidence of a myelitis is stronger in other recorded cases than it was in mine. The mention of central myelitis leads me to introduce here a notice of the one case out of 93 here collected which has not as yet received consideration. That case is the one (20) reported by Bauchet. The patient was seized whilst at dinner with trembling and sudden inability of moving her legs; at night there was complete paraplegia, and anæsthesia extended upwards to below the arms; the reflex movements of the legs were in excess; on the following day the bladder and rectum were paralysed, and on the fourth day the patient died from an upward extension of the disease. The clinical picture in this case, if not so complete as could be desired, is sufficiently so to enable us to judge with some certainty that the case was one of acute central myelitis. The lesion began in the lower dorsal region and gradually ascended, taking the muscles in the order in which they are innervated from the cord, and producing a more or less complete anæsthesia in its ascent. During the first day the lower extremities were paralysed from implication of the lateral columns, and the reflexes in the lower extremities were in excess; but as the lesion descended in the grey matter of the lumbar region, the bladder and rectum were paralysed, and had the patellar tendon reactions been then examined they would doubtless have been found lost. But be this as it may, no one can carefully examine into the course of the symptoms in Bauchet's case without being convinced that it differs greatly from the course pursued in typical cases of Landry's paralysis—and if, from the clinical evidence, we are justified in regarding Bauchet's case as being an example of acute central myelitis, we are equally justified from the same evidence in placing Landry's paralysis beyond this category, and nothing in the anatomical evidence adduced in favour of myelitis in the cases here collected is sufficiently strong to contradict this opinion.

The nerve roots or nerve trunks were found diseased in six out of the 15 cases in which positive changes were observed in the nervous system. In the case of attempted suicide by inhaling the vapour of burning charcoal, reported by Leudet (30), the patient suffered for 17 days before the onset of the paralysis, from pain and tenderness over the right sciatic nerve, and evidences of neuritis were found in the nerve after death. In Gombault's case (48), degenerated fibres were met with in some of the anterior roots and islets of sclerosis in the peripheral nerves, while in the case reported by Déjerine and Goetz (57), degenerated fibres were found in the anterior roots; and in those recorded by van der Velden (64), and Schulz and Schultze (74), in addition to the lesions in the cord, minor changes were found in the peripheral nerve trunks. In Hoffmann's case (85) degenerative changes were found in the right facial nerve, but the other nerves are said to have been normal. The most important case, however, in this respect is one of those reported by Westphal (61); the paralysis, which lasted 32 days, began four or five weeks after the patient had recovered from an attack of diphtheria, and at the autopsy the medulla oblongata and spinal cord were found normal on microscopic examination, but the anterior crural nerve showed changes similar to those which the observer had previously met with in the musculo-spiral nerve in a case of lead paralysis.

To sum up, the medulla oblongata and spinal cord were subjected to microscopical examination in 26 out of the 42 cases in which an autopsy was obtained; in 14 of these cases both were found completely normal; in four more the lesions observed were situated in the medulla oblongata and cervical region—a localisation which affords no explanation to the symptoms—and in the remaining eight cases the changes described were so slight in degree that they cannot be taken as evidence of a myelitis. Some of the nerve-roots or peripheral nerves were examined in nine only of the 42 cases, and in three of them they were found normal, while in the remaining six cases degenerated fibres and other evidences of neuritis were observed. The balance, therefore, of the evidence afforded by morbid anatomy is largely in favour of the view that the essential lesion is to be found in disease of the peripheral nerves, and nerve-roots, rather than in the nerve centres.

But the most important cases of the whole series, so far as the pathology of the disease is concerned, remain to be considered. In the case reported by Baumgarten (62), micro-organisms were found in the blood of the patient similar to those met with in the blood of a horse dead of splenic fever, the patient having been rubbed for rheumatic pains by fat supposed to have been derived from this horse. In Curschmann's case (89), Eisenlohr found typhoid bacilli scattered throughout the whole of the white substance of the spinal cord, and he communicated the characteristic disease to mice and rabbits by inoculation with these bacilli after cultivation. In Bristowe and Horsley's case (93), characteristic rabies was communicated to animals by inoculation with virus from the spinal cord. Two questions now arise with regard to these cases. Firstly, do the symptoms they present warrant their inclusion in the category of acute ascending paralysis? and secondly, granting an affirmative answer to the first question, are there any grounds for believing that these symptoms are caused by a multiple neuritis? With regard to the first question, we may at once state that in our opinion there is nothing in the course of the symptoms in these cases nor in their causation by animal poisons which will warrant their exclusion from any category of disease which embraces the acute paralysis which results from the poison of diphtheria. Regarded clinically, therefore, the cases recorded by Baumgarten, Curschmann, and Bristowe must be classed in the group of acute ascending paralysis. The answer to be given to the second question, however, must necessarily be a very hesitating one in the absence of any positive anatomical proof of the presence of a neuritis. It is, indeed, very probable that had the peripheral nerve trunks been carefully examined in these cases no morbid changes would have been discovered in them, and consequently the term neuritis or any other term implying the existence of an inflammation of a particular tissue is only applicable to them by giving a considerable latitude to our definition of inflammation. But the difficulty which meets us here encounters us also in dealing with other cases of multiple neuritis. Westphal found degenerative changes in the anterior crural nerve in a case (61) of post-diphtheritic paralysis, but in his patient the paralytic stage lasted 32 days, and it is not unlikely that had the paralysis pursued an acutely fatal course no traces of the morbid process would have been found in the nerves or elsewhere. Suppose, now, that anyone had admitted that Westphal's case, which pursued a sub-acute course, was an example of peripheral neuritis, would he be justified in denying that a case of post-diphtheritic paralysis which pursued an acutely fatal course, and in which no morbid changes could be discovered in the nerves after death, was not a case of neuritis at all? Would it not be more likely that the case which pursued an acute course is to be regarded as a neuritis equally with that which pursued a subacute one; but that while in the latter the diseased process lasted for a sufficiently long time to have permitted of such a degree of degeneration of nerve fibres to have occurred as would be readily discovered by microscopical examination, in the former a rapid effusion had taken place into the sheath of the nerves, which had the effect of arresting their functions, but not of causing changes that would be readily recognisable by our means of investigation? The truth appears to be that in rapidly fatal cases of acute ascending paralysis our present means of post-mortem investigation are not sufficiently refined to discover morbid changes, which enable us to decide whether they are to be regarded as a myelitis, a neuritis, or a toxic influence without anatomical substratum, and if preference is here given to the theory of multiple neuritis, the evidence in its favour is to be found, not in microscopical investigations, but in the essential identity of the causation and clinical phenomena of these cases with others which pursue a more or less chronic course, and in which degenerative changes have been observed in the nerves. All, indeed, that is here contended for is, that there is an essential clinical identity between acute ascending paralysis and post-diphtheritic, alcoholic, and the other forms of paralysis, which are grouped together under the name of peripheral neuritis, and that the pathology of one member of the group is, at least in a generic sense, the pathology of all. We are quite sensible that the term "peripheral neuritis" is one of Bentham's "question-begging appelatives," and feel in no way bound to adopt the theory it implies when we come to discuss the general pathology of these diseases.

In looking over the literature of this subject, whilst collecting the 93 cases here brought together, I had overlooked Nauwerck and Barth's recent contribution to the pathological anatomy of Landry's paralysis, but through the kindness of my colleague, Dr. Dreschfeld, I have now access to this important paper. Hitherto, however, I deemed it best to proceed with the analysis of my own cases, independently of extraneous aid, but now I feel free to notice the results obtained by these authors. In the first place, they have given references to several cases which I had omitted; some of these cases, like those reported by Strümpell (126), Vierordt (100), and Pitres and Vaillard (142), I had deliberately passed over, because they are admitted to be examples of peripheral multiple neuritis, and a few I had overlooked. With regard to the cases of multiple neuritis admitted by Nauwerck and Barth into the category of acute ascending paralysis, I readily allow that they have as much right of admission as many of the cases included by me, and the existence of transitional cases of this kind shows that there is no hard and fast line between the clinical group named acute ascending paralysis and the anatomical group named peripheral neuritis, and greatly strengthens the opinion that the theory of multiple neuritis is as applicable to the explanation of the former as of the latter. The authors record the case of a girl, aged 18 years, a factory worker, who began with pain in the toes and legs, and feelings of tension in the calves; soon a weakness of the lower extremities developed, and gradually increased in intensity, so that the patient, at the end of three weeks, could no longer stand without support. At the end of seven weeks from the commencement the patient was admitted to the hospital; the lower extremities were then almost completely, and the upper were partially, paralysed, and the muscles of the trunk were feeble, while the patellar-tendon reactions and the reflex actions were lost. After a time some improvement in motor power took place, but at the end of six weeks from her admission she was sitting in bed, apparently better, when she gave a loud scream, and fell on to the bed; the countenance was livid, the skin cold, the pulse scarcely perceptible, and the cardiac impulse feeble, while the pupils were dilated, the left more so than the right, and failed to react to light. The patient was unable to swallow, and soon after died from collapse. During her stay in the hospital the patient suffered for some days from severe diarrhœa, but the temperature (37·2°—37·7° C.) was practically normal.

At the autopsy the authors found great congestion and hæmorrhagic infiltration of portions of the mucous membrane of the small intestines, and on microscopical examination inflammatory infiltration of Peyer's patches. The spleen was enlarged and congested. Cheesy deposits were found in the left lung and tubercle bacilli were discovered in it, but not in the mucous membrane of the bowels, or in the spleen. A microscopical examination of the medulla oblongata and spinal cord gave negative results, but the nerves of the cauda equina, and to a less degree, the sciatic nerves, manifested a high degree of interstitial neuritis with great degeneration of nerve-fibres. Neuritic changes were also discovered in the anterior and posterior roots of the lumbar nerves, and also in greatly diminished degree in the roots of the dorsal and cervical nerves, but no changes were observed in the bulbar nerves. Tubercle bacilli were not met with in the diseased nerves. The above case, therefore, appears to have been a multiple neuritis in a tubercular subject, and it is important to observe that in the nerves first attacked—those of the lower extremities—very marked changes were observed; in those invaded next—the nerves of the upper extremities—only minor changes were noted; whilst in those attacked last—the bulbar nerves—no morbid changes could be discovered. The result, then, of the examination of the nerves in this case bears out the opinion already advanced, that in cases which pursue a rapidly fatal course it is possible that no morbid changes may be discovered even in the peripheral nerves, and if these are admitted into the category of multiple neuritis it must be, not from direct evidence, but because this theory will explain the symptoms and bring the cases under one pathology with a larger group of other cases to which they are ætiologically and clinically allied.

The following are the general conclusions at which Nauwerck and Barth have arrived:—

(1) A typical ascending paralysis, accompanied by slight sensory disorders, may prove fatal without the sphincters being implicated, or the electro-muscular irritability being diminished, while after death it may be impossible to discover any morbid change either in the central or peripheral nervous system.

(2) No sure proof has hitherto been afforded that acute ascending paralysis is in any case caused by disease of the medulla oblongata, or spinal cord, or of any part of the central nervous system.

(3) If Landry's paralysis be defined so as to include cases in which the loss of motor power is accompanied by severe sensory disorders, affections of the sphincters, diminution or loss of the electro-muscular contractility, and the reaction of degeneration, then a considerable

number of observations are recorded in which morbid lesions were observed in the peripheral nerves alone.

(4) It has not been proved that, even in the extended signification of the term, acute ascending paralysis can be caused by disease of the central nervous system.

It will be at once seen that these conclusions conform generally with the deductions already made from the preceding analysis of the cases here collected. The authors quote passages from Strümpell and Leyden, which show that they also hold essentially the same opinion with regard to the pathology of Landry's paralysis. Strümpell (126) says: "It is possible, and, indeed, many recorded facts make it even probable, that many of the cases of acute ascending paralysis belong to the group of peripheral neuritis. From these acutely fatal cases every degree of transitorial form is met with to those which pursue a chronic course." Speaking of Landry's paralysis, Leyden (159) says—"Since the observation of Eichhorst, the opinion that many, at least, of these cases really belong to the group of multiple neuritis seems to be well founded. The circumstances which speak in favour of this opinion are that acute ascending paralysis, in spite of the rarity of its occurrence, owns the same manifold ætiology as do cases of multiple neuritis; that both diseases have been observed in syphilitic subjects; that both arise spontaneously; that there is a toxic form of the one as of the other disease; and that alcoholic paralysis may appear in the form of an acute ascending paralysis."

A very full bibliography of the literature of Landry's paralysis is appended to Nauwerck and Barth's paper, and the following references are to be found in it, in addition to most of those already given in preceding pages:—

94. NAUWERCK (C.) und BARTH (W.). "Zur pathologischen Anatomie der Landry'schen Lähmung."—Beiträge zur pathologischen Anatomie und zur allgemeinin Pathologie. Von Dr. E. Ziegler. Band V., Heft 1. Jena: 1889, p. 1.
95. PICARD. *Gaz. méd. de Strasbourg*, 1867. No. 18.
96. LEIBLINGER. *Wiener med. Wochenschrift*, 1868. No. 15.
97. CHEVALET. *Bullet. gén. de Thérap.*, 1869, Oct. 15.
98. STEINER. *Arch. für Heilkunde*, 1870. No. 233.
99. KJELLBERG. *Upsala lak förhande*, 1871, VI., p. 65.
100. PETITFILS. Considérations sur l'atrophie aiguë des cellules motrices, &c. Paris: 1873.
101. BUSCH. *Berl. klin. Wochenschrift*, 1873, p. 441.
102. HENRY. Contribution à l'étude de la paralysie ascendante aiguë.—*Thèse Paris*, 1873.
103. ERB. *Arch. für Psychiatrie*, 1874. Bd. V., p. 219. "Ziemssen's Cyclopædia," 1878. Vol. XIII., p. 708.
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II. Idiopathic Peripheral Neuritis—(2) The Subacute Form.

Symptoms.—The symptoms of the subacute form of peripheral neuritis are susceptible of being divided, like the symptoms of the acute variety, into those of (1) the premonitory stage, (2) the stage of advancing paralysis, and (3) the stage of recovery. Our present purpose, however, being merely to give a general description of the symptoms as they appear in the different varieties of the affection it will suffice if we describe separately the sensory, motor, vaso-motor, secretory, trophic, and psychical disorders which are met with in the course of the disease, merely remarking that phenomena, like shooting pains and cramps, that indicate irritation of nerve tissue belong to the early, and those, like anæsthesia and paralysis, that indicate destruction of nerve tissue, belong to the later stages of the disease.

(1) *Sensory Disorders.*—Most of the cases of subacute peripheral neuritis are ushered in by the same sensory disorders as have already been described as occurring in Landry's paralysis. Probably the most common of these disorders are numbness and tingling, felt at first in the tips of the fingers and toes, but extending, as the disease advances, over the hands and up the forearms to the elbows, and over the feet and legs, as far as the knees. The patient sometimes complains that his fingers feel as if they were covered with gloves, and that it seems to him as if something soft like fur intervened between the soles of the feet and the ground, or as if his boots were filled with cold water. Patients also suffer from paroxysms of lancinating pains which shoot along the chief nerve trunks of the extremities, and these are liable to become peculiarly distressing at night, when they often assume a tearing character. These pains may become more or less fixed in one or more of the nerve trunks; at times the neuralgiform pains may assume the form of a single, or more frequently of a double sciatica, or the sensory nerves of the cervical and brachial plexuses may be implicated, while at other times the sensory nerves of the trunk are attacked and the patient then suffers from distressing pains passing like a girdle round the body.

The plantar nerves are particularly liable to be affected, and the soles of the feet are then so tender and burning as to cause much distress to the patient in walking. The course of the internal plantar nerve is very tender to pressure, and the points between the ends of the metatarsal bones, where the nerves bifurcate to form the digital branches, are particularly sensitive to touch, while another painful spot is often found about the centre of the heel, where the nerve passes through the fascia to become superficial. These distressing sensations and painful points are often accompanied by a red blush of the sole, which appears in patches of more or less extent, but the condition will be more fully described under the name of erythromelalgia. What we want to insist upon at present is that this plantar neuritis with its accompanying sensory and vaso-motor disorders is very frequently present in cases of peripheral neuritis, and forms a very marked feature of the alcoholic variety. Cutaneous hyperæsthesia is sometimes observed in peripheral neuritis, and occasionally a cutaneous nerve filament may become so swollen that it is felt like a piece of whipcord under the skin, and it is then very sensitive to pressure. The large nerve trunks, such as the musculo-spiral nerve, as it winds round the humerus, or the popliteal nerve as it passes along the external border of the popliteal space, are often found enlarged and tender to pressure. But although cutaneous hyperæsthesia is sometimes met with in peripheral neuritis, it is overshadowed by an intense hyperæsthesia of the muscles, which, in some forms, is so great as to evoke loud cries from the patient on the slightest pressure, and may at times be so extreme that the weight of the bed-clothes is intolerable, and the feet and legs have to be protected by cradles. At times the sensibility to pain may be exaggerated, while tactile sense is diminished, and in other cases there may be great diminution of the sensibility to pain, while the tactile sense is unaffected. Patients sometimes suffer from such distressing sensations of burning in the soles of the feet that they lie in bed, at least in warm weather, with uncovered feet, and the burning feet may either be dry, or bathed in a profuse and foul-smelling sweat. The hands are often similarly affected, being hot, and either dry and burning, or moist with perspiration. As the disease advances, however, the extremities become excessively cold; they may feel to the hand of the observer like the contact of a dead body, and the patient not only complains bitterly of cold hands and feet, but he has a great dread of touching anything cold, and, when possible, washes in warm water. It is difficult to know how much of these abnormal sensations is to be attributed to irritation and subsequent destruction of the nerve fibres which minister to the sense of temperature, and how much to an accompanying vaso-motor disorder to be hereafter described. Attacks of visceral neuralgia also occur in multiple neuritis. The best known form is the severe colic of lead poisoning, but similar attacks are also met with in poisoning by alcohol and other agents. A sinking feeling at the epigastrium, and even severe attacks of gastralgia are liable to occur in alcoholic poisoning, and it is probable that these and other forms of visceral neuralgia may be observed in several of the varieties of peripheral neuritis. As the disease advances every form of exaggerated sensibility gives place to diminution and ultimate loss of sensation. Diminution of sensibility is declared by retardation in the conduction of the sensation of pain or of temperature, separation of painful and tactile sensations, and Remak's double painful sensations, as well as by a diminution in the conscious reaction to painful impressions, and in the appreciation of double points. Loss of muscular sensibility is occasionally observed in peripheral neuritis; but in our experience it is only rarely present in marked degree. This point is noteworthy, for to the absence of this form of sensibility has been attributed certain disorders in the gait and in the other actions of the patient which have been thought to be of the same nature as the inco-ordinate movements of locomotor ataxia, but we have never seen any inco-ordination in peripheral neuritis in the absence of diminution or loss of motor power.

The special senses are liable to be variously affected, but consideration of these disorders is deferred until we come to describe the different varieties of multiple neuritis.

(2) *Motor Disorders.*—In the early stage of the disease the patient is liable to suffer from muscular spasms. During the active stage of the disease the affected muscles are subject to fibrillary contractions which may be seen as wavy oscillations under the skin, but do not give rise to any movement of the limb. At times the clonic spasm may be so strong as to give rise to tremor of the limbs. The trembling of the hands observed in chronic drunkards is a good example of this form of spasm, and essentially the same kind of tremor is met with in lead poisoning, and in the other forms of multiple neuritis. The tremor is usually most marked when the patient makes a voluntary movement, and in some cases the individual spasms give such a large jerk to the forearm that the hand is diverted from its course, and the object aimed at is only reached after several ineffectual attempts. Disorderly movements of this kind have been regarded as being of the nature of ataxia, but they are doubtless due to the varying degrees to which the muscles which move the limbs are implicated in the paralysis.

The peripheral segments of the limbs are most liable to be affected by tremor, but in some cases the large muscles of the proximal segments contract so powerfully as to give rise to startings of the limbs as a whole, these being peculiarly troublesome at night, and apt to occur just as the patient is falling asleep. Tonic spasms of the affected muscles are also frequent. The most usual form it assumes is cramp of the calf. This symptom is, like the startings of the limbs, apt to occur with the greatest severity ust as the patient is about to fall asleep, although he may be seized any time during the night, or on waking in the morning, or even during the day. These cramps are particularly severe and distressing in alcoholic neuritis, although they occur in other forms of the disease. The patient on being seized has generally to jump out of bed and press the toes on the floor, while he rubs first one calf and then the other with his hand, sometimes for half an hour before the spasm goes away. The fingers and toes are also liable to be variously distorted by painful cramps. Clerks, for example, find their fingers tighten over the pen, and at times the spasm may be so strong that they can neither write, nor let go their hold of the pen, until it is released by the opposite hand. Persons who perform other delicate actions, like pianists, seamstresses, and file makers, are also liable to suffer from a cramp of the fingers, which may be so severe as to render it impossible for them to carry on their respective vocations.

But the most notable phenomena of subacute peripheral neuritis are caused by a progressively advancing paralysis which usually begins, like that of the acute variety, in the peripheral segments of the limbs and gradually ascends to the muscles of the proximal joints, and finally to those of the trunk.

With regard to the *nature* of the paralysis in neuritis, it is always of the atrophic variety. The muscular masses gradually emaciate and become soft and flaccid, while no tension is provoked in them by passive movements.

The cutaneous reflexes are variously affected. In the early stage of the disease they are probably exaggerated. In some cases, strong jerkings of the limbs may be caused by tickling the soles of the feet, and the abdominal and cremasteric reflexes may be found very lively. As the disease advances, however, the reflex of the sole becomes lost, and the cremasteric reflex has also been found absent in certain forms of multiple neuritis, but the abdominal reflex is only lost in cases in which the paralysis is both profound and widely distributed.

The tendon reactions have also been found exaggerated in the early stage. We have met with exaggeration of the patellar-tendon reaction in all the varieties of the disease, but it is generally lost at a very early stage in the progress of the paralysis, and its absence is probably one of the most valuable signs of the presence of neuritis which we possess. The mechanical irritability of the affected muscle is also increased at first, but becomes lost as the atrophy advances.

The electrical reactions are variable. In the majority of cases an electrical examination of the nerves and muscles affords conclusive evidence that the paralysis of peripheral neuritis is of the atrophic kind. In aggravated cases the affected nerves and muscles lose their faradic irritability, and the muscles manifest to the galvanic current those qualitative changes which constitute the reaction of degeneration. In cases of medium intensity, however, the irritability of the nerves and muscles to the faradic current is simply lowered, but the muscles manifest to the galvanic current a reversal of the normal formula, these reactions constituting what has been called the partial reaction of degeneration. The cases in which the partial reaction of degeneration is obtained form probably by far the majority of all cases. It is very important, however, to remember that cases of undoubted multiple neuritis have been recorded in which the normal formula of galvanic reaction is maintained throughout the whole course cf the disease, and although in these cases the faradic irritability of the affected nerves and muscles may be somewhat lowered, yet a relative lowering does not enable us to form an absolute conclusion as to the nature of the paralysis. Moreover, in the early stage of the disease the irritability of the nerves and muscles to both currents may be greatly increased in degree, while there may be an absence of any qualitative change in the reactions of the muscles to the galvanic current. It will thus be seen that in the early stages of the disease, and in those cases in which the paralysis is not profound in degree, although it may be widely distributed, the reactions obtained on electrical examination tend to mislead us as to the nature of the paralysis, and consequently great caution and judgment must be exercised in drawing conclusions from the results of electrical tests.

Deformities.—The muscular wasting which occurs in peripheral neuritis produces various alterations in the contour of the body, and especially in that of the limbs. Deep furrows appear on the back of the hand, between the metacarpal bones, owing to wasting of the interossei muscles, and the space between the metacarpal bone of the thumb and index finger (Fig. 1.) becomes markedly hollow from atrophy of the abductor-indicis, adductor-pollicis, and flexor brevis muscles, while the palm of the hand loses its natural concavity, owing to flattening of the thenar and hypothenar eminences, caused by atrophy of the short muscles of the thumb and little finger. The back of the forearm is generally flattened from wasting of the extensor muscles, and the upper third of the ulnar border of the forearm loses its natural curve, owing to atrophy of the long flexors, but the radial border usually retains its normal curve, because the supinator longus remains, as a rule, comparatively unaffected. The curve of the radial border appears often, indeed, to stand out with undue prominence, from the contrast which the plump condition of the supinator longus presents to the dwindled state of the remaing muscles of the forearm. In the upper arm the skin of the posterior surface often hangs like a loose bag when the limb is held out horizontally, this appearance being caused by wasting of the triceps muscle. In most cases the deltoid, biceps, and supinator longus

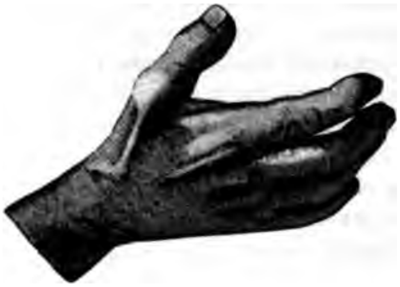

FIG. 1.—Hand from case of neuritis, showing hollow between metacarpal bones of thumb and index finger, and furrows over metacarpal interspaces of fingers.

remain comparatively free; but they also are occasionally attacked, and then the rounded prominences caused by their muscular bellies disappear, and the bones are felt almost underlying the skin. Similar changes occur in the contour of the lower extremities. The wasting of the interossei muscles and of the short muscles of the big and little toe do not give rise to such marked deformities in the contour of the foot as wasting of the corresponding muscles do in the hand; but these deformities, although smaller in degree, are seldom altogether absent, and they can be readily distinguished by an instructed observer. The prominence caused by the anterior muscles of the leg disappears, and the calf is converted into a loose bag of skin, while in advanced cases the muscular masses below the knee dwindle so much that the skin appears to lie almost on the bones. Atrophy of the muscular masses of the thigh gives rise to marked alterations in the contour of the limb, but the greatest deformity in the proximal segment of the lower extremity is caused by wasting of the gluteal muscles. The nates are flattened, the skin over the gluteal region hangs like a loose bag, and the gluteal fold feels to the observer as if it were formed of little more than a double layer of skin.

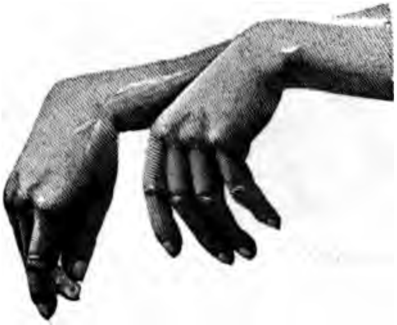

FIG. 2.—Double-wrist drop from a case of alcoholic neuritis.

But striking as are the deformities caused by wasting of the muscles, they are as nothing when compared to the vicious attitudes imposed upon the limbs and body by the unbalanced action which results from certain muscular groups being paralysed, while their antagonists are comparatively spared. The most notable feature in connection with the distribution of the paralysis in peripheral neuritis is that the extensors of the limbs are attacked in preference to the flexors. The first manifest deformities are caused by paralysis of the extensors of the forearm and of the extensors of the foot and toes, the former giving rise, when the forearms are held out horizontally in the prone position, to a double wrist-drop (Fig. 2), and the latter to a double ankle-drop (see Fig. 14). In the early stage of wrist-drop, the deformity only becomes manifest when the patient executes certain movements. When he grasps an object, for example, the action of the flexors predominates over that of the extensors, so that the hand becomes strongly flexed upon the forearm (Fig. 3). A similar flexion is observed to take place when the patient is unbuttoning or buttoning his clothes, the prominence of the curve of the flexed wrist, and the fumbling of the feeble fingers, forming very characteristic features in these actions.

Flexion of the hand at the wrist increases the distance between the origins and insertions of the extensors, and approximates the origin and insertion of the flexors of the wrist and fingers, the effect being that the extensors are stretched, while the flexors undergo adapted shortening. But the extensors, partly by their slightly remaining contractile power, and partly by their natural elasticity, offer some resistance to being stretched, and although this resistance may not be sufficient to prevent the drop at the wrist, yet it does produce extension of the basal phalanges, which are kept in a line, or almost in a line with the metacarpal bones, or may even be hyper-extended upon them. This hyper-extension is apt to occur in the flexible and delicate hands of ladies, and then a slight dimple of the skin over each metacarpo-phalangeal joint forms a feature of the deformity. The fingers are flexed at the phalangeal joints, owing to the action of the long flexors, but the action of the extensor secundi internodii causes the thumb to be extended at the phalangeal as well as at the metacarpo-phalangeal joint. The effect produced by the adapted shortening of the long flexors of the fingers becomes most apparent when the hand and forearm are held out horizontally in the supine position. The hand now falls backwards a little by its own weight, so that the tendons of the already shortened flexors are put upon the stretch, with the effect that the hand is still maintained somewhat flexed at the wrist, while the first phalanges become flexed to nearly a right angle to the metacarpal bones, the ring and little fingers being flexed to a greater degree than

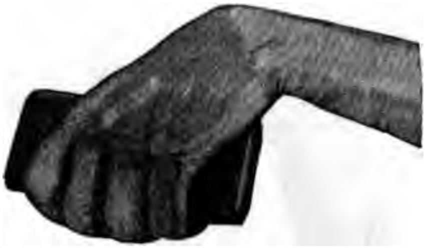

FIG. 3.—Hand from case of slight degree of alcoholic neuritis, under Dr. J. S. Bury, showing predominance of action of flexors when patient grasps.

those of the index and middle fingers (Fig. 4). The distal phalanges are, however, flexed upon one another only to a slight degree, or they may be even extended upon one another owing to the lumbricales being brought into action through the shortening of the long flexors. The position in which the hand and fingers is held is, indeed, more suggestive of an active spasm of the flexors and lumbricales than of a paralysis of the extensors.

A still further deformity occurs when the muscles of the upper arm are attacked. In these circumstances the triceps is generally paralysed to a greater degree than the deltoid or biceps, so that, in addition to the deformity already described of the hand and fingers, the forearms are maintained at right angles to the arm, and cannot be fully extended by passive movements without causing great pain, owing to the stretching which the shortened flexors have to undergo. When a patient suffering from a relative paralysis of the extensor of the upper arm as well as of those of the forearm is asked to raise his hand, the paralysed muscles fail to act, but the flexors enter into contraction, and, consequently, the forearm is drawn to an acute angle with the arm, and the hand is strongly flexed on the wrist, while the conjoined action of the long flexors of the fingers and lumbricales flex the fingers slightly at the metacarpo-phalangeal and extend them at the phalangeal joints (Fig. 5). If the pronators are paralysed to a greater degree than the supinators, the forearm is rotated so that the back of the hand is directed forwards and the tip of the fingers backwards or towards the body, and the attitude now assumed by the extremity becomes strongly suggestive of being caused by an active spasm of the flexors, the resulting distortions being very like those of tetany.

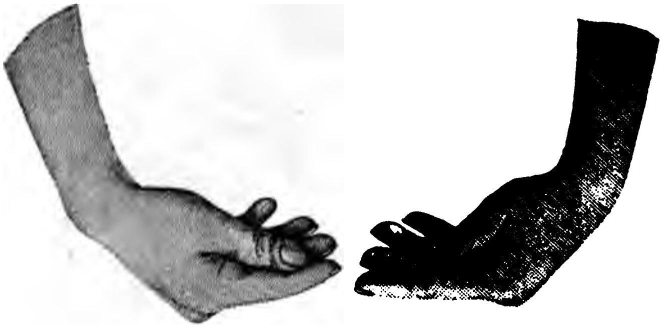

FIG. 4.—From a case of lead paralysis, in which the dropped hands were maintained in the supine position.

Besides the deformities of the fingers, hand, and forearm, caused by paralysis of the extensors of the arm and forearm, minor distortions of the fingers are caused by paralysis of the small muscles of the hand, these being of exceeding importance, inasmuch as upon a recognition of them an early diagnosis of the affection depends. Weakness of the opponens pollicis permits the metacarpal bone of the thumb to be drawn backwards by the action of the long extensors before they are completely paralysed, so that it lies on nearly the same plane as the corresponding bones of the fingers. This backward movement is accompanied by slight flexion of the distal phalanx into the palm, caused probably by weakness of the extensor secondi internodii pollicis, and a little later by a very slight flexion of the basal phalanx on the metacarpal bone (Fig. 6), caused most probably by increasing feebleness of the

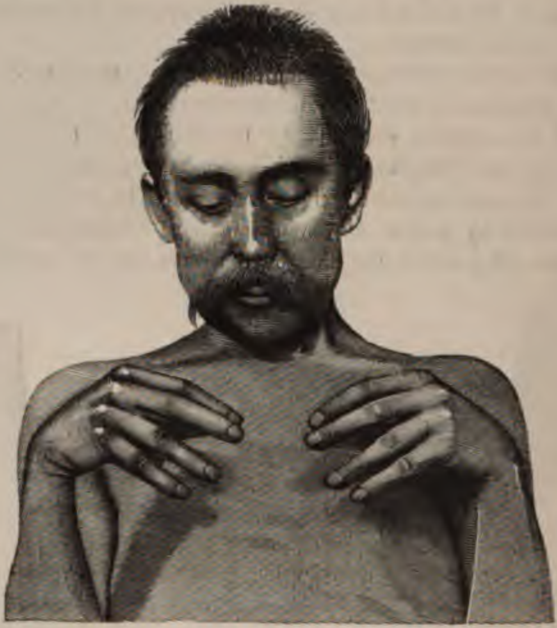

FIG. 5.—From a case of alcoholic paralysis.

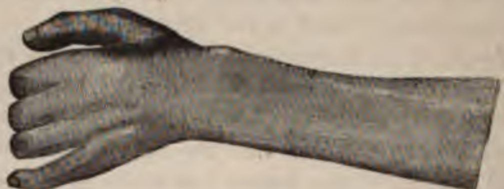

FIG. 6.—Showing metacarpal bone of thumb on nearly same plane as the corresponding bones of fingers, and slight flexion of the two phalanges of the thumb.

extensor primi internodi pollicis, while weakness of the extensor ossis metacarpi is declared by the metacarpal bone of the thumb being approximated to the metacarpal bone of the index finger (Fig. 7). The most notable distortions, however, become apparent when the patient is asked to make certain special movements with the thumb and fingers. Feebleness of the abductor brevis and opponens pollicis causes an inability of opposing the point of the thumb to the tip of the index finger, except by flexing the two last phalanges of the finger. The inner head of the flexor brevis and adductor pollicis on the one hand, and the outer head of the flexor brevis and abductor pollicis on the other, produce flexion at the metacarpo-phalangeal joint, and simultaneous extension at the phalangeal joint. Feebleness of these muscles is declared by the inability of the patient to flex the thumb into the palm without at the same time producing flexion of the distal phalanx. This disability is best made manifest by asking the patient to touch the tip of the little finger with the point of the thumb (Fig. 8), when it is found that he is unable to do so

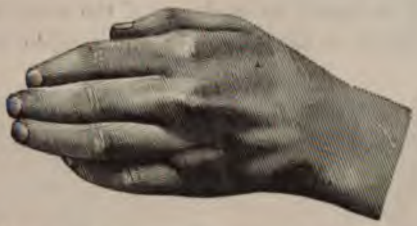

FIG. 7.—Showing adduction of metacarpal as well as slight flexion of thumb at both joints.

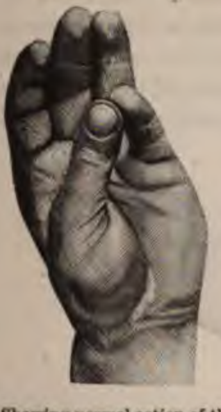

FIG. 8.—Showing normal action of thumb and little finger.

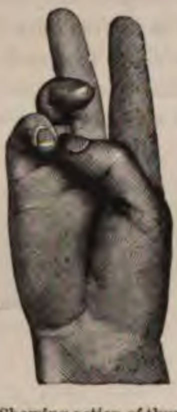

FIG. 9.—Showing action of thumb and little finger when the flexor brevis and abductor and adductor pollicis and the opponens minimi digiti are feeble.

except by flexing the distal phalanx of the thumb, and when the opponens minimi is also feeble, as generally happens, the point of the thumb touches the side, and not the tip, of the finger (Fig. 9).

Corresponding deformities to those just described as occurring in the upper extremities are also met with in the lower limbs. The first notable distortion is caused by weakness of the anterior muscles of the leg, which gives rise to an ankle-drop, in which, when the foot is unsupported, its anterior part falls downwards, so that the superior surface of the foot is nearly on the same plane as the anterior surface of the leg, while the arch of the foot is greatly increased. One effect of this distortion is to increase the distance between the origin and insertion of the extensors of the toes and to diminish that of the origin and insertions of the long flexors of the toes and of the muscles of the calf. The stretching of the extensors, which results from the ankle-drop, causes the toes to be hyperextended at the metatarso-phalangeal joints, while, by the action of the long flexors they are flexed at the phalangeal joints, with the exception of the big toe, which, in the first degree of paralysis, is hyperextended at the phalangeal as well as at the metatarso-phalangeal joint (Fig. 10). The extension of the big toe at the phalangeal joint is explained by the fact that its hyperextension at the metatarso-phalangeal joint puts upon the stretch the tendons of the abductor hallucis and inner head of the

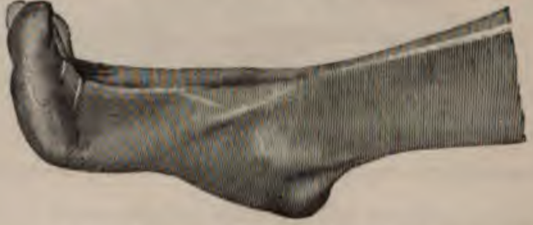

FIG. 10.—From a case of alcoholic paralysis, showing dorsum of foot on a plane nearly continuous with the anterior surface of the leg, and hyperextension of the big toe at both joints, and of the other toes at the metacarpo-phalangeal joints, with flexion at the phalangeal joints.

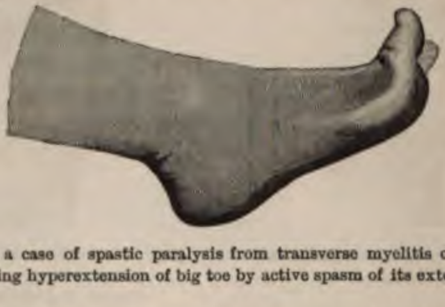

FIG. 11.—Foot from a case of spastic paralysis from transverse myelitis of the dorsal region, showing hyperextension of big toe by active spasm of its extensor.

flexor brevis hallucis, and of the adductor hallucis and outer head of the flexor brevis, and thus causes the distal phalanx to be extended upon the proximal one, the action being the same as that produced by the corresponding muscles in the thumb. This hyperextended attitude of the big toe is noteworthy, because it appears in the minor degrees of paralysis of the foot, or in the early stage of neuritis. The same attitude is met with in spastic paralysis (Fig. 11), or the paralysis which results from disease of the lateral columns of the cord, and consequently its presence in peripheral neuritis is very liable to lead to an error in diagnosis, an error into which the observer is all the more likely to fall, because in multiple neuritis, as in lateral sclerosis, the deformity may be associated with excess of the reflex of the sole, and very lively, or even exaggerated patellar-tendon reactions.

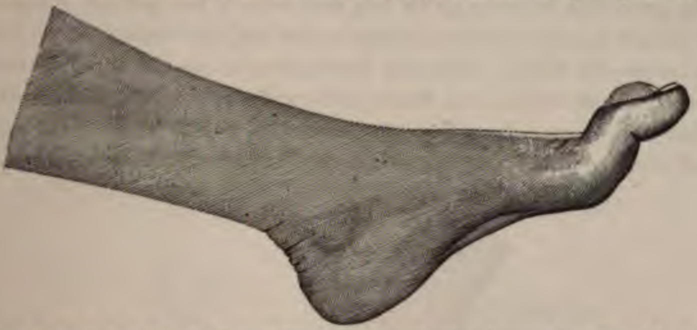

FIG. 12.—From a case of syphilitic multiple neuritis, showing hyperextension of big toe at metatarso-phalangeal and flexion at the phalangeal joint.

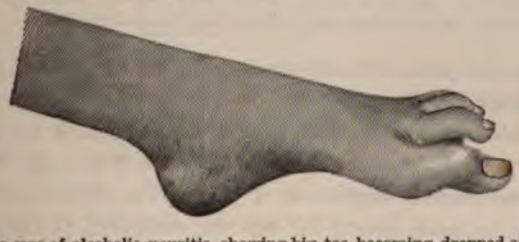

FIG. 13.—From a case of alcoholic neuritis, showing big toe becoming dropped at the metatarso-phalangeal joint, but the other toes still remain hyperextended at the corresponding joints.

In the second degree of distortion the small muscles of the big toe become too feeble to maintain the extension of the distal phalanx, and consequently it becomes flexed, but the proximal phalanx still retains its position of hyperextension upon the metatarsal bone (Fig. 12). In the third degree of distortion the proper extensor of the big toe and the first slip of the short extensor of the toes become too feeble to maintain this hyperextension and the big toe is flexed into the sole at the metatarso-phalangeal as well as at the phalangeal joint, but the little toes still remain hyperextended at the metatarso-phalangeal joints (Fig. 13). In the fourth degree of distortion, however, the little toes also are flexed at the metatarso-phalangeal joints, and the whole anterior part of the foot hangs loose and pendulous and curved towards the sole (Fig. 14.) It has been noticed that, when the foot is unsupported, its anterior part drops downwards, so that the concavity of the foot is increased in degree, but when the foot is planted on the ground it becomes quite flat, and indeed, the patient complains that his feet have become flat, or that he has lost his "spring" in walking, even before any notable deformity has occurred. In old standing cases, however, the short and long flexors have undergone adapted shortening, so that the increase in the concavity of the foot is persistently maintained, and the muscles of the sole may also undergo shortening to such a degree that the patient is unable to bring his heels to the ground in standing or walking.

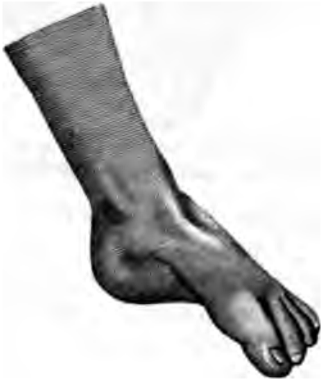

FIG. 14.—From advanced alcoholic paralysis, showing ankle-drop and pendulous condition of the anterior part of foot, with flexion of the toes at all the joints.

When the muscles of the thighs are attacked to any serious degree the patient becomes bedridden. At times he lies on his back with the various segments of the lower extremities extended upon one another, and having the superior surface of the feet almost in a continuous plane with the anterior surface of the legs, with the toes curved towards the sole. At other times the patient lies in bed with the pelvis and legs in a lateral position and the upper part of the body midway between the lateral and dorsal positions. The extensors of the leg on the thigh and of the thigh on the body being more paralysed than the corresponding flexors, the thigh becomes somewhat flexed on the body, and the legs on the thighs. Flexion at the hip and knee-joints is seldom carried beyond a right angle, but cases have been observed in which the thighs were drawn near the abdomen, and the calves were applied to the backs of the thighs. The most usual position assumed by the patient is that in which the lower extremity—say the left—rests with its outer lateral surface on the bed; the thigh is flexed to about a right angle to the body, and the leg to a right angle upon the thigh, while the foot is hyperextended at the heel and its anterior part is strongly curved towards the sole. The uppermost extremity—the right—rests on the lowermost in such a way, in the majority of cases at least, that the internal and anterior surface of the knee of the former fits into the angle formed at the internal and posterior surfaces of the tuberosities of the femur and tibia of the latter; while the leg slants towards the bed so that the angle formed by the foot at the ankle lies immediately behind the heel of the other limb, the foot, being strongly curved towards the sole like its fellow, and resting by its inner border on the bed (Fig. 15.) In some cases the right or uppermost limb rests on the left in such a way that the lower part of the thigh above the internal condyle of the femur rests on the upper part of the left leg just below the inner tuberosity of the tibia, while the lower part of the right leg above the ankle passes over the arch of the left foot, which is very concave, and the right foot, which is curved towards the sole like its fellow, rests by its inner border on the bed. The upper part of the body is turned round, so that the shoulders rest almost flat on the bed, or are only slightly inclined to the left, and the face looks upwards. After a time it is found that the muscles which have been comparatively spared have undergone adapted shortening, so that the lower limbs cannot by passive movements be fully extended at the hip and knee-joints, or flexed at the ankle, and when the patient after regaining some degree of motor power assumes the recumbent position the thighs form an obtuse angle with the body

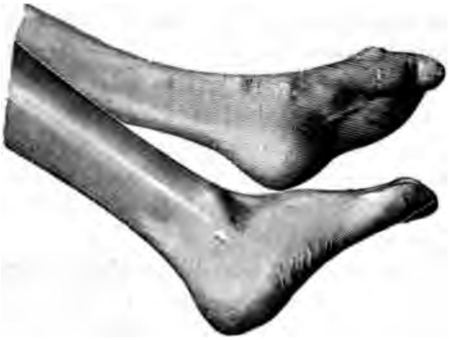

FIG. 15.—From a case of advanced alcoholic paralysis. The feet are shown as they lay flat on the bed; the right knee was uppermost, and the leg slanted backwards, so that the foot lay on the bed behind the left.

and the legs with the thighs, and thus the knees are raised while the soles of the feet rest on the bed. Feebleness of the lumbar muscles is declared when the patient assumes the erect posture. The lumbar vertebræ are curved forwards in such a way as to form a hollow in the back, and in order to compensate the incurvation of the back, the upper part of the body is thrown backwards so that a plumb-line let fall from the most prominent of the spinous processes of the dorsal vertebræ, clears the sacrum by an inch or more. When, as generally happens, the gluteal muscles are feeble at the same time, the patient experiences difficulty in rising from a chair or from the ground, and he has to assist himself by grasping his thighs above the knees just as is done in cases of pseudo-hypertrophic paralysis. It is needless to say that when cases of peripheral neuritis present this peculiar kind of disability they are liable to be mistaken for pseudo-hypertrophic paralysis.

Bilateral paralysis of the extensors of the dorsal region gives rise to paralytic kyphosis, while unilateral paralysis of these muscles gives rise to various forms of paralytic scoliosis. Weakness of the muscles of the neck is declared by the inability of the patient to raise the head or to hold it erect. Feebleness of the abdominal muscles causes impairment of the power of straining in voiding the contents of the rectum and bladder, and renders ineffective all forcible expiratory acts like coughing and sneezing, while the patient is unable to raise himself from the recumbent position without extraneous aid.

One or more of the groups of muscles supplied by the cranial nerves are frequently paralysed in this disease, but the resulting deformities and disorders of function will come under review when we come to consider the special forms of peripheral neuritis. One deformity, however, may be described here, because it may be present in most of the varieties of the disease. We allude to the peculiar expression, or, rather, loss of expression, which is caused by general feebleness of the facial muscles. The most notable effect is that all the lines which give character to the face become obliterated; the forehead loses its wrinkles, the naso-labial folds are little marked or lost, the lips are somewhat loose and flabby, and lose the fine curves which give varying expressions to the mouth, and especially the tension or compression of them, which conveys the idea of firmness and strength of character. Feebleness of the orbicular muscles of the eyelids also gives rise to a very notable change in the expression of the countenance. The lower eyelids fall to a lower level than normal, so that when the patient looks horizontally forwards, a small white belt of the sclerotic coat is seen between the cornea and edge of the lower lid of each eye. Owing to feebleness of the tensor tarsi the lower lid falls away from the eyeball, and consequently the capillary action of the lachrymal canal is lost, in some measure at least, and the tears, accumulating in the small space intervening between the everted lower lid and eyeball, give to the eye a moist and glistening appearance. The tears also flow readily over the cheeks. The tearful eye of the chronic drunkard is proverbial, but the same condition may be observed in many of the other forms of peripheral neuritis. The position of the upper eyelid also undergoes a notable alteration. The action of the levator palpebræ predominating somewhat over that of the feeble orbicular, the upper lid is drawn slightly upwards, and thus the cornea becomes all but uncovered by the upper lid, even when the patient looks forward horizontally. When the eyes are directed slightly upwards the retraction of the lid becomes still greater, so that now a narrow belt of the white sclerotic is seen between the margin of the upper lid and cornea, and the white belt already described as lying between the lower margin of the cornea and the margin of the lower lid is increased in size owing to the upward rotation of the eyeball. The consequence of the alterations in the positions of the eyelids is that the palpebral aperture is abnormally wide, and the white sclerotic surrounds the cornea on all sides, and consequently the eyeball has the appearance of being unduly prominent, as if the patient were suffering from a slight degree of exophthalmos. This retraction of the upper eyelids is generally increased when the patient comes first under observation, owing to emotional disturbance, and it gives an alarmed and frightened expression to his otherwise expressionless face.

As the disease advances the muscles of respiration are implicated. The breathing becomes accelerated, and the patient is unable to cough. If the intercostal muscles are feeble, the patient complains of a tightness across the chest, and the power of expanding the chest is found to be lost. The elevation movement, however, remains or may even be increased in degree; the extraneous muscles of respiration are then seen to contract violently, and the breathing becomes of the upper costal type. The diaphragm, however, is usually paralysed in advance of the intercostal muscles. At first, the patient complains of a feeling of oppression, and of feebleness in straining at stool or in voiding his bladder, and when the observer places his hand on the patient's abdomen during attempts at straining, he finds, either that the abdomen is not rendered tense, or that the tension can only be momentarily maintained. As the paralysis advances, the abdomen falls towards the back, so that the epigastrium is hollowed and the lower margins of the ribs become prominent (Fig. 16). The rhythm of the various movements concerned in normal breathing is now changed; the epigastrium is drawn in instead of being protruded during each inspiration, and is slightly protruded instead of falling in during expiration. Paralysis of the sphincters of the bladder and rectum have occasionally been observed in multiple neuritis, and loss of sexual power is frequently present, the impotence being on rare occasions preceded by satyriasis.

Paralysis of the muscular coat of the viscera is not unfrequently present in peripheral neuritis. It is possible that the loss of appetite and other digestive disorders which are so often observed in certain forms of the affection are due, to some extent at least, to weakness of the muscular coat of the stomach, and it is almost certain that the obstinate constipation met with in lead poisoning is caused by paralysis of the muscular coat of the bowels. It is also probable that the failure in the power of voiding the urine, which is a well known symptom of alcoholism, may, in part at least, be due to feebleness of the *detrusor vesicæ.*

But feebleness, with consequent dilatation of the walls of the heart, is by far the most important of the motor disorders of the viscera. It has long been known that in cases of diphtheritic paralysis the disease may prove fatal by syncope, the heart coming to a sudden standstill in diastole. Acute dilatation of the heart is also liable to occur in other forms of multiple neuritis, although in them death by syncope is not frequent. The patient is more often suddenly seized, during some exertion or after a heavy meal, with palpitation, dyspnœa, and other symptoms of embarrassed circulation; the face and extremities become cold, the pulse becomes frequent, irregular, and feeble or imperceptible at the wrist; there is copious vomiting; and the patient soon falls into a somnolent condition, and dies comatose some hours later. The vasomotor paralysis which accompanies the coma allows the face to become flushed, and the surface of the body to become warm, and even the pulse may acquire a deceptive fulness which may lead to the case being regarded as one of cerebral hæmorrhage, but post-mortem examinations have proved that many of these so-called apoplectic seizures—the serous apo-

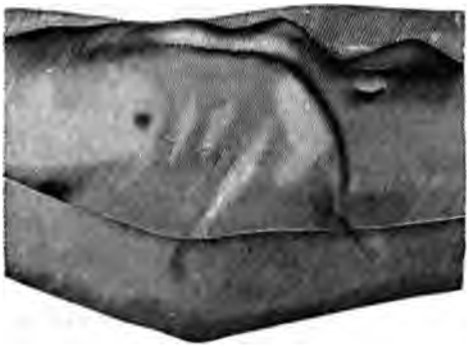

FIG. 16.—From a fatal case of alcoholic paralysis a few hours before death, showing the retraction of the epigastrium which occurs in paralysis of the diaphragm.

plexy of the old authors—are really caused by cardiac failure. In most cases, however, the cardiac dilatation pursues a subacute or chronic course, but even in these cases, the first symptoms of cardiac disease may supervene suddenly. A patient of my own, for example—a strong labouring man of 35 years of age—was ascending a hill on his way home after drinking heavily for some days, when he was suddenly seized with a severe attack of palpitation and breathlessness, and had to rest many times to take breath before he got to his home, which was only a few hundred yards away. From that time forward he was subject to attacks of dyspnœa on the slightest exertion, and a few days later his legs began to swell. At the end of ten days he was admitted as an in-patient at the Infirmary under me, and he was then suffering from anasarca and dilated heart, and although a few scattered rhonchi were heard over both lungs, it was evident, from the slight degree of bronchitis present, that the cardiac dilatation was the primary disease. In other cases, however, the distinctive cardiac symptoms come on gradually, and they often do not attract much attention until the legs begin to swell. On physical examination the impulse of the heart is found to be feeble, the apex, as determined by percussion, is displaced outwards and slightly downwards, and the transverse dulness of the heart is enlarged. The action of the heart is often irregular, and a systolic murmur may or may not be heard at the apex, but it is not conducted beyond the mid-axillary line, and is not heard at the inferior angle of the scapula, or in the vertebral groove.* The second sound at the base is often accentuated, especially if the cardiac affection is accompanied by atheroma of the arteries, and the pulmonary second sound is very accentuated or reduplicated. The veins of the neck are distended, and may sometimes be seen to pulsate. The pulse is feeble, irregular, frequent, and compressible. The pulse, and, indeed, the symptoms of the disease generally, assume a mitral character. At the same time, some of the symptoms are not unlike those of aortic regurgitation. The carotids often pulsate strongly; some of the stronger beats of the pulse give a slight jerk to the finger sufficient to remind the observer of Corrigan's pulse, and a moderate degree of capillary pulsation may be obtained by scratching the forehead. These phenomena are easily explained when it is remembered that the cavity of the left ventricle is enlarged, and that, notwithstanding the escape of part of its contents through the mitral orifice, it throws at each stroke an abnormal quantity of blood into the imperfectly distended arteries. The anasarca, which is present in cardiac dilatation, seems to me to be generally more diffused than that resulting from mitral disease. In dilatation, the walls of the chest pit readily to the pressure of the stethoscope, even when the legs are not much swollen, and the eyelids and hands are often somewhat swollen in the morning. And if along with these characteristics the urine is found to contain albumen, as not uncommonly happens, the clinical picture of the disease may be more like a renal than a cardiac dropsy. It is impossible for us, however, to pursue this subject further at present, and we shall not enter upon the wide question of the part which alcohol, lead, and other poisons take in causing atheroma of the blood vessels, and cirrhosis of the kidneys with its accompanying arterial sclerosis, or upon the secondary dilatations of the heart which may arise from these arterial degenerations.

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* *See* Steell (Graham). "Cardiac dilatation."—MEDICAL CHRONICLE, Vol. V., 1886-1887, p. 449.

Disorders of Station and Locomotion.—When the lumbar and gluteal muscles are feeble the patient experiences difficulty in attaining the erect posture, and he has, as already remarked, to aid the elevation of the trunk by grasping his thighs, or surrounding objects, just as is done in cases of pseudo-hypertrophic paralysis. In standing, the feet are held wide apart, and the upper part of the trunk is thrown back, so that the whole attitude of the patient is like that of pseudo-hypertrophic paralysis, and there can, indeed, be little doubt that some of the cases reported as examples of the myopathic disease really belong to the category of neuritis. But before the paralysis is sufficiently advanced to produce any change in the attitude of the patient on standing he finds that his feet have become flat, and that he has lost all his "spring." In the minor degrees of paralysis the loss of "spring" only becomes apparent to the patient when he attempts to run. A patient of my own discovered for the first time that he was suffering from anything more serious than the general debility which accompanies an attack of dyspepsia or other transitory ailment when, on joining a game of cricket, he found himself unable to run. Difficulty in standing on the tiptoes of one or both feet is a valuable sign of the early stage of the affection. As the paralysis advances the toes are apt to catch slight inequalities in the ground, and on entering a house the patient has to exercise great care for fear of stumbling over door-mats and rugs. His disability becomes very apparent on ascending a stair. It seems to him as if each step had become actually deeper; for he has not only to make a greater effort than usual to raise the enfeebled limb, but he has to raise the foot higher than in health in order to prevent the dropped toes from catching the edge of the step. In a further stage of the paralysis he has to assist his ascent by clutching the banister, one foot being raised first and the other dragged after.

The gait of the patient presents well marked peculiarities. The chief of these depend upon the flatness of the feet, and the drop of the toes of the advancing foot, which results from paralysis of the anterior muscles of the leg. In normal locomotion, one foot, say the right, is moved forwards, and planted on the ground in front of the other, the trunk being at the same time moved forwards and laterally, so that the line of gravity may pass through the arch of the advanced foot. The heel of the foot in rear is simultaneously raised, and now a slight contraction of the anterior muscles of the leg clears the toes from the ground, and the limb swings forward without further muscular action by a pendulum movement. When, however, the anterior muscles of the foot are paralysed, as in most cases of moderately advanced peripheral neuritis, the toes of the rear foot cannot be cleared from the ground by voluntary dorsal flexion of the foot, and as the heel is raised, the toes drop lower and lower, until their further downward movement is arrested by the anterior ligaments of the ankle-joint. One result of this drop of the toes is, that an observer, standing behind the patient, sees more of the sole of the foot at each step than he would do in the walk of a healthy person. In order to clear the toes off the ground, the heel and ankle of the advancing foot has to be unduly raised, and the necessary elevation is obtained by an unusual degree of flexion of the thigh upon the body and of the leg upon the thigh, and consequently an observer standing by the side, or in front of the patient, will note that the knee is raised to an abnormal degree, while the toes drop, the former movement impressing upon the gait that characteristic which has led Charcot to compare it to the carriage of a high-stepping horse, and the latter the appearance which has induced Schulz to compare it to the walk of a dancing-master.

When the anterior muscles of the legs are completely paralysed, the drop of the toes and the consequent elevation of the knee at each advancing step impress upon the gait such marked peculiarities as cannot but attract the attention of the most casual onlooker; but when the muscles are only partially paralysed, these peculiarities may be so slight in degree as to escape the notice of any but the most careful and instructed observer.

The above description is applicable to the kind of walk most usually observed in peripheral neuritis, but it is hardly necessary to say that the gait varies greatly according to the degree of paralysis, and according to the groups of muscles chiefly affected.

A marked modification of the above gait occurs when, as occasionally happens, the tibialis anticus muscle is comparatively spared, while the remaining muscles of the anterior group of the leg are paralysed. The heel of the foot about to be advanced being raised as in normal locomotion, the ball of the foot and with it the big toe are abruptly raised from the ground by the contraction of the tibialis muscle, while the anterior part of the outer border of the foot and the small toes drop lower and are thus the last to leave the ground and the first to touch it as the foot is being planted in front. Another important modification of the gait occurs when, in addition to paralysis of the anterior muscles of the legs, the remaining muscles of the lower extremities and back are very feeble. On standing, the patient has, as already noticed, to separate his legs widely to maintain his balance, and in order that the line of gravity may pass through the arch of the foot on the ground, the body has at each step to be drawn over to the side of that foot. In walking, therefore, the patient leans over first to one side and then to the other, so that the gait is waddling; while, to aid him in maintaining his balance, the arms are thrust out laterally like the arms of a rope-dancer. Another peculiarity of the gait in such cases is acquired from the instinctive attempt of the patient to abbreviate as much as possible the time during which the weight of the body has to be supported on one leg by taking quick and short steps. When the heel of the foot about to be advanced is raised, the foot is so quickly moved forwards that its anterior part is jerked upwards and the effects of that jerk may not have passed completely away when the foot is again brought to the ground, so that the heel may actually touch the ground first, while the toes come down immediately afterwards with a slight thump. The similarity of this modification of the paralytic gait to that observed in tabes dorsalis, has led to such cases being named pseudo-tabes. This name is, however, an unfortunate one, and ought to be rejected; and, in truth, the resemblance between the two is a superficial one, and no accurate observer who attends to the helplessness of the dependent toes, and the tottering character of the walk in cases of neuritis, can possibly mistake it for true ataxia.

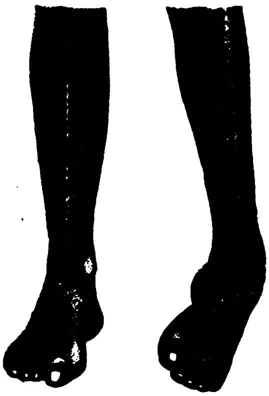

FIG. 17.—Showing position of feet in a case of neuritis in which the tibialis anticus muscle on each side is comparatively spared. The left figure shows the position of the foot when it is firmly planted on the ground, while the right shows the position it assumes just as the patient is making a voluntary effort to raise the foot.

Vasomotor and Secretory Disorders.—In the early stages of peripheral neuritis the vessels of the surface, and especially those of the palms of the hands and soles of the feet, are liable to become dilated. The affected part then assumes a bright, or at times, a dusky red tint, the surface temperature is raised, and the patient complains that the palms of the hands and soles of the feet are hot and burning. As the disease advances the kind of passive hyperæmia and lowered temperature, which Raynaud has called "local asphyxia," is apt to occur, and then the extremities feel cold and clammy to the touch, and the patient complains bitterly of cold hands and feet. In some cases the hands become quite pale and bloodless on being held vertically, and of a dark purple tint on being held in a dependent position. Many alcoholic patients, indeed, find that their hands readily swell, and become of a dark red tint, when they are held in a dependent position, and the feet are liable to be similarly affected, the soles becoming, when the feet are dependent, of a dark purple colour. In the early stages of the disease, the tendency of the hands and feet is probably to be hot, burning, and dry; but as the disease advances, they become warm and moist, and at a still later period, cold and clammy. while the sweat in which the feet are bathed is apt to be particularly foul-smelling and offensive. The temperature of the body remains normal throughout the course of the disease, unless there is a complication of pneumonia or other acute disorder. A moderate rise of temperature may also be present in the terminal period of fatal cases when the muscles of respiration are becoming progressively paralysed.

Trophic Disorders.—The atrophy which the paralysed muscles undergo has been already described, and it only remains for us now to allude briefly to the remaining trophic disorders met with in the course of the disease. The nutrition of the skin suffers in various ways, but the most usual effects are that it becomes thin and loses its wrinkles, while the surface becomes smooth and glazed or glossy, these changes being particularly liable to occur over the palms of the hands and backs of the fingers. Bed sores have occasionally been observed, but they are of rare occurrence, and the profound destructive changes met with in acute myelitis are probably never present in neuritis. The nails frequently undergo nutritive changes; they become dry, furrowed longitudinally, and so brittle that they readily crack and become jagged at their edges. At other times the nutritive changes occur chiefly at the matrix of the nail, and then the attached end or the whole nail becomes furrowed and deformed. The joints, especially the small joints of the fingers, and more rarely of the toes, are liable to be swollen and tender in multiple neuritis, and quite independently of any rheumatic or gouty taint. Into the question of whether the joint affections of gout and rheumatic gout may not themselves be the result of a neuritis we do not enter. Perforating ulcer, with disease of the subjacent bone, may be caused by inflammation of peripheral nerves, but it has only been occasionally observed in cases which come under the category of uncomplicated multiple neuritis.[*] The subcutaneous tissue of the palm of the hand sometimes undergoes a kind of hypertrophy or hyperplasia,[†] and when this is accompanied, as generally happens, with wasting of the muscles of the thenar and hypothenar eminences, the palm of the hand becomes quite flat, while its surface loses all its lines and wrinkles, and becomes smooth and glossy, these last appearances being doubtless caused in part at least by nutritive changes in the skin itself. In cases of wrist-drop from lead or even alcoholic poisoning, a painless swelling may appear over the wrist-joints, which is caused by a swelling, probably of an inflammatory nature of the extensor tendon. This condition has been described under the name of *tenositis hyperplastica*,[‡] and is by some regarded as a trophic lesion, and by others as resulting from the mechanical irritation to which the stretched tendons are exposed while running over the strongly-flexed wrist. But whatever may be the origin of this condition, it has the effect both of increasing the deformity during the stage of paralysis, and of retarding recovery, inasmuch as the swollen tendons may, by forming adhesions with their sheaths, prevent the hands from regaining their normal positions, even after motor power has been restored.

Psychical Disorders.—The psychical disorders of peripheral neuritis are both numerous and important, but inasmuch as they do not occur, at least in any marked degree, in every form of the disease, consideration of them will be deferred until we come to describe the individual varieties.

III.—THE CHRONIC FORM.

The chronic form of multiple neuritis is generally a sequel of the acute or sub-acute varieties. In three out of the cases of acute ascending paralysis here collected the paralysis supervened more or less acutely, but improvement was slow, and the distortion of the extremities remained more or less persistent. In one of these cases (40) the paralysis reached its maximum of intensity and distribution in two or three weeks, but the *main en griffe* persisted at the end of two years; in another (50) the maximum degree of paralysis was reached in seven days, but the patient was unable to stand at the end of twelve months, while the movements of the upper extremities were feeble; and in the third (48) the paralysis reached its maximum in five days, but deformities of the extremities were noted seven years later, when the patient died of abdominal cancer, and islets of sclerosis were then found post mortem in the peripheral nerves. Had any of these patients been seen for the first time at the date of the last report of their cases it is very probable that the disease would have been regarded, at least a few years ago, as a progressive muscular atrophy in which the wasting of the muscles had become stationary for a time, and yet the history of two of these cases at least (40 and 50) clearly shows that the paralysis was caused by alcoholic excesses. In hospital practice it is not all uncommon to meet with cases of chronic paralysis, with wasting of some of the muscular groups of the extremities, and consequent deformities of the feet and hands, and in which the origin of the disease is somewhat difficult to trace. A few weeks ago a woman was under my care, in the Infirmary, suffering from double ankle drop, slight distortion of the toes, general wasting of the muscles of the legs, absence of the patellar tendon reflexes, and marked dimunition of the faradic contractility of the nerves and muscles of the legs and feet. There were, however, no marked cutaneous sensory disorders, the muscles were not tender to pressure; the patient did not suffer from cramps, and the hands were unaffected; in one word, what we may call the *aleoholic stamp* was completely wanting. On enquiring into the history of this patient, however, there was no difficulty in proving that the paralysis was of alcoholic origin. She made no secret of the fact that up to 18 months ago she drank to great excess; she then suffered from shooting pains in the extremities, cramps of the calf, great tenderness of the whole body, and became almost completely helpless and bedridden; and to make assurance doubly sure, she was at that time an inmate of the Infirmary for many weeks, and Dr. Reynolds, medical superintendent of the Infirmary, informed me that she was then suffering from alcoholic paralysis in its most typical form. Another Infirmary patient of mine was the subject of double-ankle and double-wrist drop, but without marked sensory disorders. On being cross-examined as to her habits, she admitted in a very shame-faced manner that she had taken liquors to excess, but it turned out that she had once taken rather more than was conducive to good behaviour on the occasion of a public festivity, and had consequently fallen into disgrace, but she was in no way an habitual tippler. The origin of her case was cleared up when a small blue line was discovered on the gums. This line disappeared about a week after her admission into the Infirmary, and, consequently, had she come under observation a week or ten days later than she did, it would have been impossible to have ascertained the origin of the paralysis, and the case would have been regarded as a multiple neuritis of unknown origin.

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[*] See Helbing (P.). "Ueber perforirende Hautgeschwüre in Folge von Neuritis."—Aus der Tübinger chirurgischen Klinik des Professor Dr. Bruns. Beitrage zur *klinischen Chirurgie*, Bd. II., 1889. Abstr. *Centralbl. für Nervenheilkunde.* Leipzig: 1889. Jahrg. XII., p. 722.

[†] See Löwenfeld. "Zwei Fälle neuritischer Platthand."—*Münchener med. Wochenschrift*, 1889, No. 24. Abstr. *Centralbl. f. d. med. Wissenschaften*, Berlin, 1889, p. 880.

[‡] See Erb (W.). *Ziemssen's Cyclopædia*, Vol. XI., 1876, p. 550.

A case which well illustrates the difficulty of tracing the disease to its origin came under my observation a few years ago. Mrs. M., aged thirty-eight years, consulted me on June 24th, 1884. She resided abroad, but paid an annual visit to her friends in this country. When in this country in the summer of 1883, she felt weak and "out of sorts," but was so much benefited by the change of air that she was able to return to her distant home much improved in health. Soon afterwards, however, she began to complain of a sensation of "fidgets" in her left leg, accompanied by shooting pains, as if electricity were passing through it. Her right leg was soon afterwards attacked, and she felt herself day by day becoming weaker at the knees. In three or four weeks from the commencement her lower extremities were almost completely paralysed. After a time her hands were invaded, but it was not until the Christmas of the same year that she observed that her hands were becoming notably wasted and distorted. The patient and her husband stated that she was unusually abstemious in her habits, and only partook of a single glass of claret at dinner, by her doctor's orders.

Present Condition.—On walking across the floor it is seen that the toes drop with each advancing step, and that the knee has to be elevated to an unusual degree in order to clear the toes off the ground. When the patient is seated on a chair, and the feet are placed flat on the ground, she is unable to raise the ball of either foot from the ground by voluntary effort. In the left upper extremity the thenar and hypothenar eminences are wasted; the hand is distorted into the form of a claw, and the grasp is feeble. The extensors of the forearm are also weak, but not sufficiently so to give rise to a manifest wrist-drop. The right upper extremity is similarly affected, but in a much less degree than the left. The affected nerves and muscles react to a faradic current of moderate strength, and the muscles react in a normal manner to galvanic stimulation. There are no sensory disorders, either subjectively or to objective examination. The patellar tendon reactions are absent. The patient is being skilfully treated by her husband, who has been carefully instructed in the method of applying galvanism by her foreign physician.

This patient did not again come under my observation until two years later, when the following report of her case was taken:—May 30th, 1886.—Mrs. M. called to-day, and I find that her general health is excellent, and that she has regained almost completely the motor power of her lower extremities. The nerves and muscles of the lower limbs give normal reactions to electrical stimulation; the patient's gait is quite normal; she can now raise the ball of the toes of either foot from the ground by voluntary effort, but the patellar tendon reactions are still absent. The left hand is distorted into the form of a claw, and the right is also affected, although it is not so much deformed as the left. The muscles of the thenar and hypothenar eminences do not react to the strongest faradic or galvanic current. The flexors of the forearm give a feeble reaction to a galvanic current from 35 cells Leclanché on anodal and to 40 cells on cathodal closure. The muscles of the shoulders and upper arms are somewhat wasted and feeble. The face is smooth and expressionless, and the palpebral aperture of the right side is wider than that of the left. The patient can still close her eyes, but the slightest pressure of the observer's thumb suffices to overcome the contraction of the orbicular, the action of that of the right side being especially feeble. The pupil of the right eye is wider than that of the left, but the two react both to light and accommodation. The external rectus of the right eye appears to be feebler than the corresponding muscle of the left eye, as judged by the power of the respective muscles to rotate the eyeball outwards, but the patient does not complain of double images. The patient states that she occasionally experiences some difficulty of articulation, but at her visit she pronounced polysyllabic words without difficulty or perceptible disability. The sense of temperature is completely lost in the left hand, but the other forms of cutaneous sensibility are not much affected, and the patient does not complain of numbness or other paræsthesia. The muscles are not tender to pressure.

August 30.—Mrs. M. presented herself again to-day for examination. For the last week she has been suffering from digestive disorders, accompanied by diarrhœa. In repect of her paralytic condition her symptoms have continued more or less unchanged. She now complains of a feeling of oppression, and of a sensation of constriction around the chest. The difference in the circumference of her chest, on a level with the nipples, between her maximum expiration and inspiration, is two inches, and the diaphragm acts forcibly. I now prescribed for the diarrhœa and digestive disorders, but have not seen or heard of the patient since that time.

The notes of the above case are somewhat imperfect, but the most careful enquiries were made into all circumstances which would tend to connect the symptoms with the action of the poisons of syphilis, diphtheria, alcohol, and lead, but with negative results; and although I have the strongest conviction that the case was an example of peripheral neuritis, I am totally unable to conjecture what was the nature of the poison which caused it.

In other cases the disease may extend over many years, but its progress is interrupted by alternate amendments and relapses. The following is a brief report of the symptoms in a case of what I may call relapsing paralysis, which came under my observation some years ago, and which I then regarded as being a probable example of periependymal myelitis.

Ann R., aged 33 years, unmarried, entered the Manchester Royal Infirmary under my care on May 25, 1882. The patient's occupation was "cotton-balling," an operation which consists in winding cotton into a ball of considerable size. She enjoyed excellent health until seven or eight years previously, when she began to feel her fingers awkward and feeble when at work, while at the same time she observed that the little and ring fingers dropped powerless when the hands were held out horizontally in the prone position. She suffered from severe pain between the shoulders, but from no other sensory disturbance. The weakness of her hands, however, increased rapidly, and she was soon obliged to desist from work; but after a rest of three weeks' duration she resumed her occupation, and continued steadily at work for the next three years. At the end of that time she was seized with some kind of fit. Paralysis of both hands developed suddenly, and six or seven months elapsed before she regained full motor power in them. During the four years before she came under observation her history has been that whenever she worked for some time her hands became more or less suddenly paralysed, and on resting gradual recovery took place in three or four months, to be followed by a relapse soon after she resumed work. For the last two years she has not worked more than a few weeks at a time when the paralysis reappeared, and lately a few days at work sufficed to induce an attack.

Present Condition.—The patient is well nourished and of healthy appearance, except that her eyelids are a little puffy, but all her internal organs are normal; there is no albumen in the urine, and no blue line on the gums. The patient has double wrist-drop, her grasp is feeble, the thenar and hypothenar eminences are flattened, and she is unable to perform special movements with her fingers, while the hands have a characteristic claw appearance. The pronators and supinators of the forearm and the muscles of the upper arm are unaffected. The extensors of the forearm and the opponens and abductor pollicis do not react to the strongest faradic current; but the long flexors of the forearm, the flexor brevis, and adductor pollicis, and the muscles of the hypothenar eminence react feebly to a current of medium strength. The muscles which failed to react to the faradic current react to a galvanic current from 15 cells Leclanché on anodal closure, and with 30 cells on cathodal closure, while those which contracted to a medium faradic current react to a galvanic current from about 25 to 35 cells on anodal, and an equal number on cathodal closure. The patient states that her finger tips are numb, and that her hands are burning; but there is no diminution of sensation to objective examination. She also complains of tingling sensations in her feet, but the muscles of the lower extremities do not give evidence of paralysis, and all of them give normal reaction to electrical examination. On July 5, 1882, the patient was discharged, and became an out-patient.

February 1, 1883.—Considerable improvement has taken place in the patient's power of extending the hands and fingers, but her grasp is still feeble. She now complains, however, of numbness of her feet, and of a constrictive pain surrounding her body on a level with the false ribs, and her feet have "lost all their spring." On examining the lower extremities, it is found that the anterior muscles of the right leg and the intrinsic muscles of the foot are almost completely paralysed, while those of the calf only contract feebly on voluntary effort. The corresponding muscles of the left leg are similarly affected, but in less degree. The affected muscles of the right leg fail to react to the strongest faradic current; but those of the left leg give a very feeble reaction to a very strong current. The muscles of the right leg contract to a galvanic current from 30 cells on cathodal and 25 on anodal closure, while those of the left leg contract to 35 cells on cathodal and an equal number on anodal closure. The feet and outer surfaces of both legs are relatively anæsthetic, and the patient cannot localise touch clearly over these parts.

The patient continued to improve whilst she remained under treatment, but had not recovered completely when she disappeared from observation.

During the time that the patient, whose case has just been reported, was under my care, her sister was being treated for a somewhat similar affection by my colleague, Dr. Dreschfeld.* The patient was admitted as an in-patient of the Infirmary, under Dr. Simpson, on February 14, 1882. She was 43 years of age, and was suffering from paralysis and atrophy of the upper extremities. She had been married 23 years, and had twelve children, nine of whom are living. Her occupation was that of a "cotton-baller," but she had not worked at the mill since her marriage. Three weeks before her admission she felt a sharp pain, which was especially severe over the little and ring fingers, shoot across her right hand, and soon afterwards her wrist dropped, and her arm also became partially paralysed, the movements at the shoulder being more feeble than those at the elbow. Fourteen days later the left arm was similarly attacked with shooting pains; it also soon became more or less paralysed. The patient suffered from great pain in the affected extremities, and also from pain in the back, which shot across from shoulder to shoulder.

Present Condition.—On examination, Dr. Dreschfeld found the patient suffering from marked paralysis, and atrophy of the muscles of the fingers and hand, and of the extensors of the fingers and wrist, and to a less degree of the flexors of the fingers and wrist. The triceps was also weak and atrophied, and the deltoid, biceps, and supinator longus were somewhat feeble. The patient complained of numbness and coldness of her fingers, but there was no manifest anæsthesia. The affected muscles gave degenerative reactions on electrical examination. The patient came under Dr. Dreschfeld as an out-patient, in May, 1882; he then found that she had improved considerably, and ultimately she made a complete recovery.

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* Dreschfeld (J.). "On some of the rarer forms of muscular atrophies."—*Brain*, Vol. IX. London, 1887, p. 187.

In January, 1884, she again presented herself suffering from pains, loss of motor power, and muscular atrophy, with degenerative electrical reactions in the upper extremities, and after a few months' treatment again made a good recovery. In March, 1885, she appeared for the third time as an out-patient, suffering from what proved to be a comparatively mild attack, and from which she made a quick recovery.

With respect to the cause of the neuritis in these two sisters it is difficult to form a trustworthy opinion. Knowing that they were more or less similarly affected by paralysis, and that both were by occupation "cotton-ballers," I suspected that there was some inherited weakness in the spinal cord, which was excited to actual disease by over-exercise of the muscles of the upper extremities induced by the nature of their work, but I abandoned this theory on hearing that Dr. Dreschfeld's patient had not worked at "cotton-balling" during the 23 years of her married life which had preceded the first attack.

I am now inclined to believe that the disease must have been caused in both by chronic poisoning of some kind, and I cannot think of anything that would be more likely to have induced their symptoms than secret drinking. When, however, these patients were under observation, our knowledge of multiple neuritis was much less accurate and definite than it now is, and no very stringent enquiries were made into their habits. But whatever may have been the cause of the disease in these two patients, the truths we should like to enforce at present are, that cases come under our observation in which a widely distributed paralysis, with atrophy, has existed for years, either in a continuous or in a relapsing form; that the association of pains and other sensory disorders with the paralysis, in the early stage of many of these cases, prove them to have been caused by disease in the course of the nerve trunks; and that the exciting cause may have disappeared so completely as to render its nature a matter only of conjecture. Many of the cases of progressive muscular atrophy, reported by the older authors,* seem to us to belong to the category of chronic peripheral neuritis, in which the nature of the exciting cause has not been traced. We shall now proceed to consider the special varieties of peripheral neuritis, and shall first describe the form resulting from the poisonous action of the diffusible stimulants.

* See Friedreich (N.). *Ueber progressive Muskelatrophie*, Fall I., p. 11; Fall VI., p. 31. Berlin: 1873.

III. TOXIC MULTIPLE NEURITIS—(1) THE NEURITIS CAUSED BY DIFFUSIBLE STIMULANTS.

(*a*) ALCOHOLIC PARALYSIS.

WHEN it is considered that the habit of drinking fermented liquors has been prevalent amongst all advanced races from the earliest period of their history, and that the habitual indulgence to excess in these beverages gives rise to a very remarkable and striking series of disabilities and distortions of the body, it can hardly be doubted that the symptoms resulting from chronic drinking must have attracted the attention of instructed observers at an early period in the history of medicine. It is at least certain that the tremors, the progressive emaciation, the insecure and staggering gait, the numbness and loss of feeling in the extremities, the distortions of the fingers, and the chronic indigestion which result from excessive indulgence in wine, were known to Seneca,[*] and were the search likely to prove profitable, it is hardly to be doubted that more or less accurate descriptions of these symptoms would be found in the writings of Hippocrates and Galen and of the Arabian physicians. Coming down to more modern times, we find that the famous Dr. Lettsom[†] gave, just a century ago, a vivid and accurate description both of the insidious manner in which the drinking habit is often acquired, and of the paralysis which is the natural result of it.

The passage is so important that we need not offer any apology to our readers for quoting it in full. After describing *first* the symptoms of the chronic indigestion of hard drinkers and, *second*, the symptoms which precede and attend cirrhosis of the liver, the author proceeds: "The *third* train of symptoms to be described is not confined to age or sex, but is in general more frequently the attendant of the female sex. The persons liable to these symptoms have been those of delicate habits, who have endeavoured to overcome the nervous debility by the aid of spirits; many of these have begun the use of these poisons from persuasion of their utility, rather than from the love of them; the relief, however, being temporary, to keep up their effects frequent access is had to the same delusion, till at length what was taken by compulsion gains attachment, and a little drop of brandy or gin and water becomes as necessary as food; the female sex, from natural delicacy, acquire this custom by slow degrees, and the poison being admitted in small doses, is slow in its operations, but not less painful in its effects.

"The soberer class of tradesmen also, who occasionally indulge in their sixpennyworth of brandy and water, gradually slide into the same unhappy habits, and entail upon their constitutions the same misery which I shall now introduce.

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[*] Seneca. *Epist.* 93, § 16. Quoted by Brühl-Cramer. "Uber die Trinksucht und eine rationelle Heilmethode derselben."—Berlin, 1819, p. 53.

[†] Lettsom (J. C.). "History of some of the effects of hard drinking." London, 1789, p. 5, *et seq.*

"The first appearance of indisposition very much resembles what has been last described: and under the deception of the gout, the fuel is heaped upon the fire, till the delusion has been too long maintained to admit of retreat: in general, at least, the attachment to the use of spirituous drinks becomes so predominant, that neither threats nor persuasions are powerful enough to overcome it. The miserable sufferer is so infatuated, as, in spite of locks and keys, to bribe by high rewards the dependent nurse, privately to procure. But the concluding symptoms are very different from either of the foregoing histories; frequently, indeed, the appetite for food vanishes, but sometimes continues voracious; and, at the same time, whilst the body is costive, and no vomiting ensues, the lower extremities grow more and more emaciated; the legs become as smooth as polished ivory, and the soles of the feet even glossy and shining, and at the same time so tender, that the weight of the finger excites shrieks and moaning; and yet I have known, that in a moment's time, heavy pressure has given no uneasiness. The legs, and the whole lower extremities, lose all power of action; wherever they are placed, there they remain till moved again by the attendant; the arms and hands acquire the same palsied state, and the patients are rendered incapable of feeding themselves. Thus for years they exist, with no material alteration in the size of the body or aspect of the countenance.

"Whether they really undergo the agonies they appear to suffer I much doubt, as at this period their minds appear idiotish; they often shriek out with a vehemence that may be heard at a considerable distance, but upon enquiring about the seat of pain, they have been vague and indecisive in their answers. When a cramp comes in the lower extremities, involuntary motions draw up the legs, and produce the most piercing shrieks, and the features of the face, altered by convulsive twitchings, excite pain in a spectator. For some months before they die their shrieks are more incessant, and as violent as the strength will admit. They talk freely in the intervals of mitigation, but of things that do not exist; they describe the presence of their friends as if they saw realities, and reason tolerably clear upon false premises."

The above passage requires no comment. The extreme hyperæsthesia, the emaciation, the glossy skin, the hysterical outcry, with distortion of the face on being touched, the utter helplessness of the lower extremities, and the peculiar kind of dementia observed in advanced cases of alcoholic paralysis, especially as observed in females, are here so graphically delineated that it cannot but be a subject for marvel how it has happened that the profession, once put in possession of such a powerful and accurate description, had ever afterwards lost sight of it, or had overlooked the causal connection existing between certain forms of paralysis and alcoholic excesses.

The truth appears to be that the physician continued to regard these cases as belonging to the province of the alienist, and that the alienist, approaching the subject from his own standpoint, looked upon them as forming one of the varieties of general paralysis of the insane, a disease which is often complicated at its onset by alcoholic excesses, but which often attacks individuals who have not been exposed to the action of this poison, or to any other obvious and exciting cause. From this point of view it might be possible to regard the alcoholic excesses as merely forming part of the extravagances which are so common during the stage of exaltation, and not as the true cause of the disease.

The symptoms of alcoholic paralysis, with its accompanying sensory and vaso-motor disorders, were again described with great fidelity in 1819 by Brühl-Cramer,* who, from his position as practising physician in Moscow, had excellent opportunities of observing the deleterious effects produced by the abuse of raw spirits.

A still more accurate and detailed description of these symptoms was given in 1822 by Dr. James Jackson, a physician, of Boston, who appears to have been the first to have directed attention to the predominance of paralysis of the extensors over that of the flexors. This description bears internal evidence of having been drawn from nature, and is a model of accurate observation, but as it has been quoted in full by my colleague, Dr. Dreschfeld,† it is not necessary for me to reproduce it here.

But, important as have been the descriptions of alcoholic paralysis given by the above authors, it is unquestionable that to Dr. Magnus Huss belongs the merit of being the first to have given a minute and detailed account of the symptoms from an elaborate analysis of a considerable number of carefully recorded cases. He was the first, so far as we know, who pointed out the similarity of alcoholic paralysis to that resulting from chronic poisoning by lead, copper, mercury, and other metals, and from ergot. The work was, indeed, so thoroughly done by Huss that very little has been added since his time to our knowledge of the clinical history of the disease, if we except our present information with regard to the state of the tendon and electrical reactions, but the value of these, as signs of disease, was hardly known in his day.

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* *Loc. cit.*, p. 53.

† Jackson (Dr. James). "On a peculiar disease resulting from the use of ardent spirits." *New England Journal of Medicine and Surgery*, Vol. XI. (third series, Vol. I., No. 4), Boston, 1822, p. 351. Quoted by Dreschfeld (Julius). "Further observations on alcoholic paralysis."—*Brain*, January No., Vol. VIII., London, 1886, p. 433.

The fact that alcoholic excesses give rise to a particular form of paraplegia was pointed out many years ago by Dr. Wilks,[1] and contributions to the subject were made by Dr. Handfield Jones,[2] Mr. Lockhart Clarke,[3] and Dr. Reginald Thompson,[4] while Leyden[5] also stated that he had seen several cases of transitory paralysis from alcohol, and Westphal[6] described a case of peculiar kind of gait arising from the same cause. Cases of alcoholic paralysis were reported by Topinard,[7] Bourdon,[8] and others, as examples of locomotor ataxia, and Jaccoud[9] mentions chronic alcoholism amongst the causes of tabes, so that he also must have mistaken the uncertain and staggering gait of alcoholic paralysis for the true ataxic gait.

The sensory disorders caused by the abuse of alcohol were specially studied by Leudet,[10] and the cases reported under the name of general neuralgia[11] appear to belong to this category.

The next great advance in the study of alcoholic poisoning was taken by Lancereaux,[12] who was the first to suggest that the symptoms were caused by inflammation of the peripheral nerves, and also directed attention to symptoms like local asphyxia and gangrene of the extremities, which had hitherto been overlooked. Very important contributions to the subject now followed in quick succession by Myrtle,[13] Glynn,[14]

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[1] Wilks (S.). *The Medical Times and Gazette*, Vol. II., 1868, p. 470. "Alcoholic paraplegia." *The Lancet*, Vol. I., 1872, p. 320, and "Lectures on diseases of the nervous system," 2nd Edit., 1883, p. 372.

[2] Jones (C. Handfield). "Notes on epileptic attacks and paralysis as results of alcoholic excess."—*The Practitioner*, Vol. VII., London, 1871, p. 331.

[3] Clarke (J. Lockhart). "Alcoholic paresis and paraplegia."—*The Lancet*, Vol. I., Lond. 1872, p. 427.

[4] Thompson (Reginald). "On paralysis of the extensors."—*Medico-Chirurgical Transactions*, Lond. 1868.

[5] Leyden (E.). "Klinik der Rückenmarkskrankheiten." Berlin, 1875, Bd. II., Abth. I., p. 281.

[6] Westphal (G.). "Ueber eine bei chronischen alcoholisten beobach. Form von Gehstörung." *Charité-Annalen*, Berl. 1877, p. 395.

[7] Topinard. "De l'ataxie locomotrice." Paris, 1864, p. 41.

[8] Bourdon (H.). "Études cliniques et histologiques sur l'ataxie locomotrice progressive."—*Arch. génér. de Méd.*, Vol. II., 1861, p. 515.

[9] Jaccoud. "Les paraplegies et l'ataxie du mouvement." Paris, 1864, p. 630.

[10] Leudet (E.). "Etudes clinique de la forme hyperesthésiques de l'alcoolisme chronique et de sa relation avec les maladies de la moelle."—*Arch. génér. de Méd.*, 6th Série, Tome IX., 1867, p. 5.

[11] See Valleix. *Bullet. de Thérapeutique*, Tome XXXIV., 1848, Obs. 2, 3, and 4. *Gazette des Hôpit.*, 6th March, 1849, and 21st September, 1850; and Leclerc, "De la Névralgie Généralé," *Thèse de Paris*, 1852, Obs. 2, 4, 5.

[12] Lancereaux (E.) Art. "Alcoolisme (Pathologie)."—*Dict. Encycl. des Sciences Méd.*, Tome II., Paris, 1865, p. 615 *et seq.* "De la Paralysie Alcoholique."—*Gazette Hebdom. de Méd.*, 2 Série, Tome XVII., Paris, 1881, pp. 119, 165, and 195. "Des troubles vaso-moteurs et trophiques liés à l'Alcoolisme et à quelques autres intoxications chroniques (paleurs, sueurs, froids, asphyxie locales oedème et gangrene des extrémites.")—*L'Union Medical*, Tome LXIII., Paris, 1831, p. 745; and "De l'absinthisme chronique."—*Gaz. Med. de Paris*, 1881, pp. 14, 15, 21, 23, 24.

[13] Myrtle (A. S.) *The British Medical Journal*, Vol. II., 1880, p. 312.

[14] Glynn (T. R.) "Cases of Alcoholic Paraplegia."—*The Liverpool Medico-Chirurgical Journal*, July, 1883, p. 374.

Broadbent,[1] Dreschfeld,[2] and Hadden[3] in this country; by Fischer,[4] Möeli,[5] Löwenfeld[6] in Germany; by Charcot and Féré,[7] and Déjerine[8] in France; and by Hun[9] in America. Numerous other contributions to the subject have been made in recent years, and of these we may mention papers by Krücke,[10] Strümpell,[11] Lilienfeld,[12] Schulz,[13] Lancereaux,[14] Bernhardt,[15] Standish,[16] Kast,[17] Oppenheim,[18] Saundby,[19] Findlay,[20] Duckworth,[21]

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[1] Broadbent (W. H.) "On Alcoholic Poisoning."—*The Lancet*, Feb. 16, 1884, p. 294.

[2] Dreschfeld (J.) "On Alcoholic Paralysis."—*Brain*, Vol. VII., July, 1884, p. 200, and "Further Observations on Alcoholic Paralysis."—*Brain*, Vol. VIII., 1886, p. 433.

[3] Hadden. "Alcoholic Paralysis."—*British Med. Journal*, Vol. II., 1884, p. 818, and *Transactions of the Pathological Society*, London, 1885, p. 49.

[4] Fischer (G.) "Eine eigenthümliche Spinalerkrankung bei Trinkern."—*Archiv für Psychiatrie*, Bd. XIII., Berlin, 1882, p. 1.

[5] Möeli. [*Berl. klin. Wochenschrift*, 1884, p. 223.] "Statistisches und Klinisches ueber Alcoholismus."—*Charité Annalen*, Berlin, 1884, p. 541, *et seq.*

[6] Löwenfeld (L.) "Ueber Spinallahmung mit Ataxie."—*Archiv für Psychiatrie*, Bd. XV., Berlin, 1884, p. 438.

[7] Charcot and Féré. "Des paralysies alcooliques."—*Le Progrès Médical*, June 14, Paris, 1884, p. 475.

[8] Déjerine (J.). "Étude sur le nervo-tabes périphérique (ataxie locomotrice par névrites périphériques, avec intégrité absolue des racine postèrieure, des ganglions spinaux, et de la moelle epinière."—*Archives de Physiologie normal et Pathologique*, Serié III., Tome III., Paris, 1884, p. 231.

[9] Hun (Henry). "Alcoholic paralysis."—*American Journal of Med. Sciences*, 1885, April, p. 372.

[10] Krücke (Dr.). "Die Pseudotabes der Alcoholiker."—*Deutsche Medizinal Zeitung*, 1884, No. 72.

[11] Strumpell (A.). "Ueber die Nervenerkrankung der Alcoholisten."—*Berl. klin. Wochenschrift*, 1885, No. 32.

[12] Lilienfeld (A.).—*Neurolog. Centralbl.*, Jahrg. IV., Leipz., 1885, p. 352: and *Berl. klin. Wochenschr.*, 1885, No. 45.

[13] Schulz (R.). "Beitrag zur Lehre der Multiplen Neuritis bei Potatoren."—*Neurolog. Centralbl.*, Jahrg. IV., 1885, pp. 433, 462, 482.

[14] Lanceréaux (E.). "Paralysies toxiques et paralysies alcooliques."—*L'Union Med.*, Paris: 1885, No. 32.

[15] Bernhardt (M.). "Ueber die multiple Neuritis der Alkoholisten; Beiträge zur differentiellen Diagnostik dieses Leidens von der Tabes, der Poliomyelitis subacuta, und der sogenannter Landry'schen paralyse."—*Zeitschrift für klin. Medicin*, Band XI., 1885, Heft IV.

[16] Standish (Myles). "A case of alcoholic paralysis preceded and accompanied by amblyopia ex abusu."—*Boston Medical and Surgical Journal*, 1886, April 22.

[17] Kast (A.). "Klinisches und Anatomisches über primäre degenerative Neuritis."—*Deutsches Archiv f. klin. Med.*, Bd. XL., 1886, Heft 1.

[18] Oppenheim (H.). "Beiträge zur Pathologie der 'multiplen Neuritis' und Alkohol-Lahmung."—*Zeitschrift für klin. Med.*, Berl., 1886, p. 232.

[19] Saundby (R.). "A case of alcoholic paraplegia."—*The Lancet*, Vol. II., 1886, p. 241.

[20] Findlay (D. W.). "Three cases of alcoholic paralysis (multiple neuritis)."—*Medico-Chirurgical Transactions*, London, 1887.

[21] Duckworth (Sir Dyce). "Three cases of multiple peripheral (alcoholic) neuritis in women."—*St. Bartholomew's Hospital Reports*, Vol. XXII.

Smart,[1] Buzzard,[2] Witkowski,[3] Thomsen,[4] Déjerine,[5] Uhthoff,[6] Sharkey,[7] Taylor,[8] Bramwell,[9] Siemerling,[10] Drysdale,[11] Tiling,[12] Korssakow,[13] and others.

In addition to these papers several important theses and monographs have been published on the subject. Without pretending to give an exhaustive list of them we may mention the works of Oetlinger,[14] Leval-Picquecheff,[15] Béhague,[16] and Korssakow,[17] while it is hardly necessary to remind the reader that much information is obtained in alcoholic paralysis in papers and monographs, such as that of Dr. Buzzard, on the general subject of peripheral neuritis.

We now propose to illustrate the course and symptoms of alcoholic paralysis by describing a few typical cases of the disease. For the records of these cases we are chiefly indebted to Dr. W. R. Williamson, Medical Registrar to the Infirmary, who superintended and corrected the notes of the clinical clerks.

Case 1.—Jane H., æt. 36 years, was admitted to the Royal Infirmary on January 6th, 1890, under the care of Dr. Ross. The patient is a married woman, but has had no children and no miscarriages. Her husband has been engaged for the last two years in making furniture polish, and there is always a strong smell of naphtha in the house. At the same time she admits that for many years she has taken a considerable quantity of whiskey and other stimulants, and for the last eighteen months she was obliged to take a glass of spirits at night to procure sleep and relieve the pain caused by "rheumatics" in the legs. The patient suffered at various times from numbness and tingling of the hands and feet, and from cramps in the legs, but her memory is defective, and it is doubtful how far her statements can be depended upon. She states that she experienced weakness of the legs for the first time about a month ago, and that she lost the use of her hands somewhat suddenly, about a week before her admission, but her statements with regard to dates must be accepted with caution.

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[1] Smart (A.). "A case of multiple neuritis."—*Edinburgh Medical Journal*, July, 1888.

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Present Condition.—The patient is a stout woman, of florid complexion and bloated countenance. All her muscles are flabby, but the presence of a considerable quantity of subcutaneous fat prevents the appearance of much wasting, except in the muscles of the calves, which are manifestly atrophied. There is a little pitting, on pressure, over the ankles. As the patient lies on her back in bed the lower extremities are flaccid and powerless, while the feet are dropped at the ankles and the toes at all the joints. The patient is unable, as she lies on her back, to move the lower extremities in the slightest degree at any of the joints. When placed on her side, however, she can produce slight flexion of the legs at the knees, and of the thighs at the hips, but without being able to extend them. In short, with the exception of this slight power of flexion, the legs remain wherever they are laid until moved by the attendant. When the forearms are held up vertically the hands are completely dropped at the wrists and the fingers at the metacarpo-phalangeal joints, and the patient is quite unable to make the slightest movement of extension of the hands or fingers. The grasp of either hand is feeble and the patient cannot oppose the tip of the thumb to those of the fingers, and cannot separate the fingers from one another. Extension of the forearms at the elbows is relatively feeble but the other movements at the elbow and

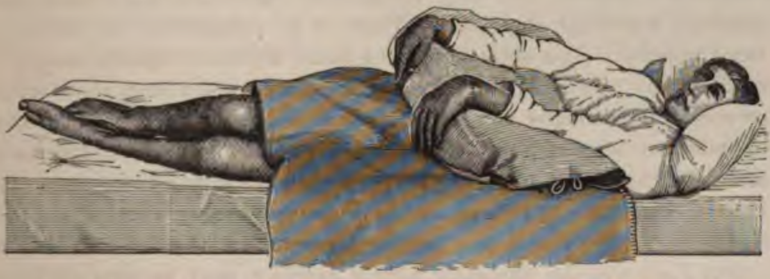

FIG. 18.—From a photograph of Jane H., as she lies in bed with her legs laid on their sides.

shoulder joints are performed with considerable force. The reflexes of the sole, the patellar-tendon reactions, and the wrist jerks, are absent; and the abdominal and epigastric reflexes are also feeble or absent. The patient complains of rheumatic pains in the forearms and about the shoulders, and of numbness and tingling of the fingers and toes, and in the backs of the hands and feet. The head of a pin is not perceived on the backs of the hands and fingers, or on the dorsal surfaces of the toes, but the prick of a pin is perceived in those parts, although the sensibility is somewhat delayed. She can tell the position of the legs, and the direction given to them by passive movements when the eyes are closed. The soles of the feet, especially along the course of the plantar nerves, are very sensitive to pressure, and the slightest pressure over the calves of the legs and over the muscular masses generally, evokes loud outcries of pain. There is nystagmus of both eyes when the patient tries to fix an object, and the oscillatory movements become very marked when the eyes are directed to lateral objects. The pupils are moderately dilated, but they contract to light and accommodation, although somewhat sluggishly. There is no paralysis of the ocular or facial muscles. The optic discs were found normal on ophthalmoscopic examination. There is no central scotoma, and vision is good in other respects. The tibialis anticus reacts on either side to a faradic current of medium strength, but the extensor longus digitorum and peronei muscles fail to react to strong currents. In the upper extremities the exterior communis digitorum reacts to a very strong current on the right side and to one of medium strength on the left, while the extensor ulnaris, the supinator longus, extensor ossis metacarpi, flexor sublimis, flexor radialis, and flexor ulnaris muscles in both arms react to weak currents.

The following reactions were obtained to the galvanic current:—

	Left Leg.		Right Leg.	
	KSC.	ASC.	KSC.	ASC.
Tibialis anticus.........Cells Leclanché	10	15	10	10
Extensor longus digitorum	20	20	15	10
Peronei	15	20	15	15

	Left Arm.		Right Arm.	
	KSC.	ASC.	KSC.	ASC.
Extensor communis digitorum	20	20	15	15
Extensor ulnaris	15	15	10	15
Supinator longus	20	20	20	25
Extensor ossis metacarpi	15	15	15	20
Flexor sublimis	20	25	25	25
Flexor ulnaris	15	20	20	20
Flexor radialis	20	20	20	20

Nothing abnormal is discovered in the heart, lungs, or digestive organs.

The urine is 1,024 in specific gravity, and free from albumen. The temperature is normal.

It is found that the patient's memory is very defective, and that she is particularly apt to make contradictory statements with regard to dates. At one time, for example, she stated that she was an in-patient in the Infirmary eighteen months ago, and on the following morning that she was an in-patient six months ago. On closing her eyes for sleep she has often seen faces, and at other times figures of real persons standing by her bedside. She has also been much troubled with horrid dreams, and recently she has seen numbers of cats coming about her bed.

January 28.—This patient is still in the Infirmary, undergoing treatment by massage and electricity, but as yet, there is no perceptible improvement in motor power.

Case 2.—C. W., aged 21 years, was admitted to the Royal Infirmary on October 23rd, 1889, under the care of Dr. Ross. The patient became a sailor at a young age, and for the last six years has been a steward on board a trading vessel. He always enjoyed good health until six years ago, when he had an attack of rheumatic fever, for which he had to keep his bed for about a month. He made a good recovery, however, and remained in good health until three years ago, when he had an attack of sunstroke in Bombay. His father died at 33 years of age from consumption, but his mother is now alive and healthy. He has also a brother and sister living, and enjoying good health. The patient states that for the last five weeks he has been gradually becoming weaker, and during this time he has lost 10lbs. in weight. Six weeks ago he could lift a weight of 2 cwts. with ease, but now he is unable to lift a chair with one hand, and on making the slightest effort his body becomes bathed in profuse perspiration. He also perspires very freely both night and day independently of any exertion. About three weeks ago he noticed for the first time that he had considerable difficulty in raising his feet on ascending a stair, and after making the effort the calves of his legs felt very sensitive and painful. He also suffered from numbness and tingling of the hands and feet, while the soles of the feet were very sensitive to pressure. The patient has suffered for the last fifteen months from cramps of the calves. He was most usually seized soon after getting into bed, but some of the attacks have come on as he awoke in the morning. The cramps have been particularly frequent, severe, and painful for the last few weeks, and during an attack he has had to jump out of bed and press his feet against the cold floor, while sometimes he has had to walk about the floor for over half an hour before the spasm completely subsided. The patient states that his sleep has been disturbed for some time by dreams, but they were not of a particularly disagreeable character. His mind has been occupied by dreamy fancies of his being the possessor of great wealth, but he laughingly refuses to enter upon a more detailed description of them. On closing his eyes for sleep at night he has for the last few weeks seen coloured clouds before his eyes, in the midst of which faces appear, just like photographs upon a coloured screen. These faces were generally pleasing, and he has never seen animals like cats or rats about his bed. He never heard any voices. On being questioned as to his habits, he states that his daily allowance of spirits was four glasses of rum, but as he had the keys of the stores, he had the opportunity of taking more if he liked, but he is somewhat reticent as to the quantity he took in addition to the regulation four glasses.

Present Condition.—The lower part of the patient's face is somewhat ruddy, and slightly sunburnt in appearance, but his forehead, temples, and eyelids have a pale and sallow complexion, while scratching the forehead with the nail brings out a slight degree of capillary pulsation. The carotids beat visibly, and the jugular veins are dilated but do not pulsate. The lower extremities and the surface of the trunk generally pit slightly on pressure.

The patient complains of numbness and tingling of the fingers and toes, but there are no manifest cutaneous sensory disorders to objective examination. The calves of the legs and the muscular masses generally are exquisitively sensitive to the slightest pressure. The most accessible nerve trunks, such as the ulnar and the external popliteal, are also tender to deep pressure. The internal plantar nerves are extremely sensitive to pressure along their whole course, and are particularly tender between the heads of the metatarsal bones where the branches bifurcate to form the digital nerves. When the feet are dependent the soles become of a dark violet colour, this tint being most marked on the inferior surfaces of the toes and anterior part of the sole. When the patient sits on the side of the bed he experiences some difficulty in attaining the erect position, and has to assist himself by grasping his thighs. On standing, his feet are held wide apart, and he totters, and in walking there is a well marked drop of the toes with each advancing step. As the patient lies on his back in bed, a slight degree of double ankle-drop is observed, but there is no manifest distortion of the toes. His grasp is feeble, and he is unable to touch the tips of the little finger and thumb, without flexing the distal upon the proximal phalanx of the thumb. The reflex of the sole is very lively, and the patellar tendon reaction is exaggerated on both sides. The cremasteric reflex is sluggish, but the abdominal and epigastric reflexes are active. The electrical reactions were not examined. The apex of the heart is displaced somewhat outwards and the transverse cardiac dulness is enlarged; a soft systolic murmur is heard over the apex, but it is replaced by an impure first sound at mid-axilla and is not heard at the back. The second sound, at the base, is accentuated. The pulse beats 72 in the minute and is regular, moderately full and very compressible. The urine is acid, the specific gravity is 1015, and it is free from both sugar and albumen. His temperature is normal. On reading for a little time the print becomes blurred and his eyes dim, so that he is obliged to desist, but the fundus of each eye is found normal on ophthalmoscopic examination. The hallucinations of sight have ceased since he entered the Infirmary, and he sleeps soundly. He was ordered 10 minims of tincture of digitalis in an ounce of diuretic mixture three times a day.

November 9.—The œdema disappeared from the ankles in a few days and the patient rapidly improved in strength, but although he was discharged, yet he had by no means recovered the full motor power of his hands and lower extremities.

Case 3.—H. H., aged 28 years, entered the Manchester Royal Infirmary, under Dr. Ross, on December 9th, 1889.

The patient is unmarried, has been a day labourer, and much exposed to changes of weather. He states that for many months he has been most temperate, only averaging about two glasses of beer a day. A few weeks ago he began to suffer from general weakness, palpitation, and breathlessness, and soon afterwards he observed that his ankles were swollen at night. These symptoms were not preceded by shooting pains or attacks of cramp, but as he became gradually weaker, he sought admission as an in-patient at the Infirmary.

Present Condition.—The patient has a bloated appearance and watery eye, which is suggestive of chronic alcoholism. On standing, his feet are wide apart and he totters; he is unable to stand on his tip-toes, and in walking there is a slight drop of the toes with each advancing step. As he lies in bed his feet manifest a slight degree of double ankle drop, but beyond a slight degree of hyperextension at the metatarsophalangeal joints, there are no distortions of the toes. All the movements of the lower extremities can be voluntarily performed as the patient lies in bed, but dorsal flexion of each foot is feeble and imperfect. The grasp is feeble, but there is no wrist-drop, and the ordinary special movements of the thumbs and fingers can be performed, although with manifest effort and with some accompanying tremor. The patellar tendon reactions are lost, but the cutaneous reflexes are maintained.

There are no cutaneous sensory disorders to objective examination, but the patient states that for a long time his hands and feet are very sensitive to cold, and when they are unduly exposed they become deadly cold and numb. The muscles of the calves of the legs and of those of the forearm are slightly tender to deep pressure; so also are the branches of the internal plantar nerve.

The lower extremities and scrotum are œdematous, and the mark of the stethoscope is readily left on the front of the chest during examination of the heart and lungs. The apex of the heart is displaced outwards slightly beyond the nipple line, and the transverse dulness on a level with the fourth rib is somewhat enlarged. A soft systolic murmur is heard over the apex, but it is replaced at mid-axilla by an impure first sound, and is not heard at the back. The second sound at the base is accentuated, but the sounds at the base are free from murmur. The carotids beat visibly, and a slight degree of capillary pulsation is obtained on scratching the forehead. The pulse at the wrist beats 88 in the minute; it is somewhat jerky in character, slightly irregular, and very compressible. The jugular veins are distended, but do not pulsate.

The urine is free from sugar and albumen, and the temperature is normal. An electrical examination shows that the muscles of the lower extremities all react to a moderate faradic current. The following are the galvanic reactions obtained.—

		Right Leg.		Left Leg.	
		K.S.C.	A.S.C.	K.S.C.	A.S.C.
Tibialis anticus	Cells Leclanché	25	30	25	30
Peronei	,, ,,	25	30	30	35
Extensor longus digitorum		25	30	30	30

At the first examination of this patient two circumstances in his case seemed very puzzling. The first one was, that the quantity of beer which he consumed, according to his own statement, seemed hardly likely to have produced even the slight degree of paralysis which he presented, and still less likely to have caused the cardiac dilatation and consequent anasarca; and the other was, that granting his case to be one of alcoholic paralysis, it seemed strange that the loss of motor power was not preceded by the usual tearing pains and cramps of the lower extremities. On being further pressed with regard to his habits, however, it turned out that, 18 months ago, he had been drinking beer very heavily, and he then suffered from a paralysis, which was preceded by the most excruciating pains and cramps in the lower extremities. His body and legs were swelled as at present, and he was for many weeks an inmate of the Infirmary. This statement was confirmed by Dr. Reynolds, the medical superintendent, who remembers that his case was one of typical and very aggravated alcoholic paralysis. Under treatment by digitalis and diuretics the œdema disappeared in a few days, and at the end of a fortnight he was, by his own desire, discharged. It is not likely, however, that the heart is thoroughly reconstituted, and under the strain of the heavy work of a labourer, it is highly probable that the patient will soon break down again, even if he abstains from alcohol.

Case 4.—J. C., aged 42 years, entered the Royal Infirmary under Dr. Ross September 9th, 1889. The patient is a brewer's drayman by occupation, and has been much exposed to cold and wet during the winter months. He has always lived in a healthy and comfortable house, and been warmly clad. He enjoyed good health up to four years ago, when he had an illness very similar to his present attack, and which was also accompanied by swelling of the lower extremities and body. He made an excellent recovery then. He remained in comparatively good health from that time till the beginning of the present seizure, except that for the last eighteen months he has suffered, on getting up in the morning, from severe attacks of coughing, which were accompanied by much retching, and occasionally by vomiting, but by little or no expectoration. He has also suffered from numbness and tingling and great coldness of the hands and feet, and has been greatly distressed by severe attacks of cramp of the calves of the legs, which usually came on soon after he had gone to bed. He is married, but his wife has had no living children; she had one dead-born child and two miscarriages. His father is living and healthy, but his mother died some years ago from heart disease. He has had ten brothers and sisters, but only four are living now, three having died from consumption, one from heart disease, and two in childhood. The patient states that he rarely takes more than two or three pints of beer a day. On being cross-examined, however, it is found that by this statement he means that he seldom pays for more, but in addition to the above quantity he takes his regular allowance and what he gets from others. His regular allowance consists of a glass of beer for every barrel he delivers, and he admits that these may number from nine to ten a day. At public-houses he is often treated to an extra glass. He also acknowledges that he gets "proper drunk" regularly once a fortnight.

Present Condition.—The patient is a stout man with bloated countenance, and a capacious barrel-shaped chest. The lower extremities and trunk are œdematous, and the backs of the hands also pit slightly on pressure. He complains of numbness and coldness of the hands and feet, and of shooting pains in the lower extremities, but there are no cutaneous sensory disorders to objective examination. The muscular masses of the lower and upper extremities are extremely sensitive to pressure, and so also are the accessible nerve trunks—the plantar nerves being particularly sensitive. The patient's grasp is feeble, and he cannot perform certain special movements with his fingers, but there is no manifest wrist drop. As he lies on his back in bed there is a slight indication of double ankle drop, but the toes are not distorted. The movements of the lower extremities are feeble, but none of them are quite lost. The reflexes of the sole are lively, and the abdominal and epigastric reflexes are maintained; but the cremasteric reflex is sluggish, and the patellar tendon re-actions are lost. The muscles of the abdomen, as tested by asking him to raise himself in bed when in the recumbent position, are very feeble, and the abdomen is not rendered very tense when the patient is asked to strain, although the diaphragm is not completely paralysed. The circumference of the chest on a level with the nipples is 44 inches, but there is only a difference of one half inch between his maximum inspiration and maximum expiration. The chest is very resonant over the front and back, but loud rhonchi are heard widely diffused over both lungs. The expectoration consists of a frothy mucus, and is only moderate in quantity. The respirations are 24 in the minute, and of a somewhat upper costal type. The apex of the heart is displaced a little downwards and outwards, and the transverse deep dulness is enlarged. The pulse is 120, irregular, feeble, and very compressible. The jugular veins are distended, but there is no pulsation of them. The carotids, however, pulsate visibly, and a very slight degree of capillary pulsation is observed on scratching the forehead. The first sound at the apex is impure, and the second at the base accentuated, but there is no murmur. The urine is high-coloured, specific gravity 1,018, and contains a trace of albumen. The temperature is 98° F. The pupils are contracted, but re-act to light and accommodation. He was ordered an expectorant mixture, to each dose of which 10 minims of tincture of digitalis was added.

Sept. 13.—Since the date of last report the patient became steadily worse. For the last two nights he has been wandering, and has passed his urine in bed, but the temperature has never risen above normal. His breathing has become increasingly difficult, and he died during the day from asphyxia. No autopsy was obtained.

Case 5.—J. C., aged 42 years, was admitted on June 18th, 1889, to the Manchester Royal Infirmary, under the care of Dr. Ross. The patient's mental condition is such that he is unable to give a connected history of his antecedents, but it has been ascertained, from his friends, that his occupation is that of a greengrocer, who goes about from house to house with a small cart, selling fruit and vegetables. His father, three brothers, and two sisters, are still living, but his mother died some years ago. The only fact that can be ascertained about his personal history, beyond his occupation, is that he has been a noted drunkard.

Present Condition.—The patient is fairly well nourished, but his face is smooth and expressionless, the palpebral fissures are unduly open, and a belt of fluid is apt to collect between the globes and the margin of the lower eyelids. The patient does not complain of numbness or coldness of the hands or feet, and there are no cutaneous sensory disorders to objective examination. The muscles of the calves of the legs, and those of the forearm are markedly tender to pressure. The patient has double wrist drop, and is unable to perform certain special movements with his fingers and thumb. The feet are also dropped at the ankles, and when he walks there is a marked drop of the toes with each advancing step, giving to the gait a well-marked high stepping action. The reflexes of the sole are active, but the patellar-tendon re-actions are absent. His urine is free from albumen. The temperature is 98°F. The pulse is rather frequent, being generally about 90 in the minute, but besides accentuation of the second sound of the heart at the base, there are no cardiac symptoms deserving of note. The pupils are somewhat dilated, the right being slightly wider than the left, and they are sluggish to light and accommodation; the ocular movements are normal.

The patient's mind is almost a complete blank. He cannot tell anything about his father and brothers and sisters, and it is impossible to find out from him whether he has ever been married or not.

July 11.—The patient has remained in the same condition as at his admission. His bodily health is good, his temperature has remained throughout normal, but he has not regained additional motor power, and his mind is in a state of complete imbecility. He has been kept in bed until the afternoon of each day since his admission, but at the physician's visit he announces that he has just returned from a long walk, gives a detailed and circumstantial account of having met with and spoken to a friend, of having called at certain public-houses, and of having had several glasses of beer. His walks were supposed to be taken in Didsbury, this being the district in which he hawked his vegetables. The following is a sample of his usual statements. On being asked, "Have you been out walking this morning?" "Oh, yes. I had a walk out yonder." "Did you meet any one you know?" "Yes; I met a friend, and him and me went into the public-house up yonder—I forget the name—and had a glass of beer." "Was it the Blue Bell?"—a wrong name being purposely suggested. "Yes," he replies dubiously; then proceeds more airily, "it is the public-house at the corner up yonder. You know it quite well. We had a glass of beer there." "And had you any more than one glass?" "Well, yes; we then came down to that other public-house, and had another glass there." A somewhat similar conversation took place day by day at the physician's visit, and all this from a man who had been in bed for the greater part of each day for a month, and had not been outside the ward during that time. As no improvement took place in the bodily or mental condition of the patient he was now discharged.

For the notes of the following case I am indebted to Dr. Williamson, Medical Registrar to the Royal Infirmary: —

Case 6.—P. J., aged 54 years, was admitted to the Royal Infirmary on August 3rd, 1889, under the care of Dr. Ross. He has been married for many years, but has had no family. He is a pork butcher by trade, and has been much exposed to wet and cold, but until recently he has always enjoyed excellent health. He admits that he has consumed daily large quantities of beer and spirits.

About a month ago the patient noticed, for the first time, that his ankles were swollen at night, and that his face and eyelids were puffy in the morning. For some considerable time he has suffered from shooting pains in the legs, numbness of the fingers and toes, and deadly coldness of the hands and feet, more especially in getting up in the morning. In previous years his hands and feet were warm, and in summer weather his feet became so hot and burning that he had to leave them uncovered at night. For the last twelve or eighteen months, however, his feet are always cold, and he has been unable to get them warm by any means whatever. He has at various times suffered from attacks of cramp, which usually seized him in one or both calves, just as he was dozing off to sleep. At times the cramp was so severe that he had to jump out of bed and press his toes on the ground, but on most occasions it subsided when he rubbed the calf for some time as he sat in bed. He was also subject to attacks of cramp in his fingers, and occasionally the fingers of the right hand closed so tightly round the handle of his knife that he could not let go his hold until the knife was released by the aid of the other hand.

Present Condition.—As the patient lies in bed his countenance has a bloated and besotted appearance and stupid expression; the eyelids are puffy, the palpebral fissures are widely open, and a rim of fluid collects along the margin of the lower lid on each side, giving to the eyes a tearful look. His lower extremities, the buttock and the back of the trunk, are swollen and pit on pressure, and the mark of the stethoscope is left on the surface of the chest during an examination of the lungs and heart.

The grasp of the patient is feeble, and although he can perform all the movements of the lower extremities whilst lying down, he is quite unable to stand without support. When the arms are held out horizontally in the prone position the patient can by voluntary effort extend the hands and fingers, but a slight drop of each hand at the wrist is observed when he moves the upper extremities without paying particular attention to the position of the hand, and also when he is made to perform special movements like buttoning and unbuttoning his shirt. He is also unable to touch the tips of the little finger and thumb without flexing the distal phalanx of the thumb upon the proximal one. As he lies on his back in bed, with the lower extremities extended, the feet are seen to be somewhat dropped at the ankles, but the toes are hyper-extended at the metatarso-phalangeal and flexed at the phalangeal joints, except the big toe, which is extended at the phalangeal joint as well. The reflex of the sole and the patellar-tendon re-actions are much exaggerated on both sides. The cremasteric reflex is sluggish, but the abdominal and epigastric reflexes are normal.

Cutaneous sensibility, as tested by the pricking of a pin, is much diminished over the feet and legs, and the appreciation of pain and of heat is delayed, by a few seconds, over these parts. The soles of the feet are tender to the slightest touch, pressure over the internal plantar nerves and their branches being excessively painful. The muscles of the body generally are exquisitely tender to pressure, and the slightest squeeze of the muscles of the calf evokes loud cries, followed by an almost hysterical outburst of tears. The patient is deaf, hearing the ticking of a watch only at 3 inches from the left and 1½ inches from the right ear. On examination of the chest there is no abnormal dulness on percussion, but scattered rhonchi are heard over both lungs. The apex of the heart, as determined by percussion, is situated in the sixth interspace, 2½ inches below and 1 inch to the outside of the left mammary line. The transverse dulness on a level with the fourth rib begins 1 inch to the right of the right edge of the sternum, and extends to the outside of the left nipple a distance of 6¾ inches.

The first sound at the apex is impure, and the second sound at the base is somewhat accentuated, but there is no murmur. The carotids beat visibly, the jugular veins are distended and pulsate slightly, and a faint capillary pulsation is seen on scratching the forehead. The pulse is 110, slightly irregular, and compressible. The tongue is furred, the bowels are constipated, and there is great loss of appetite. The urine, when voided, is acid, the specific gravity is 1,018, and it contains a slight trace of albumen. For the last few weeks he has been very sleepless, and has been much troubled at night by seeing faces making horrible grimaces at him, as well as rats and other animals creeping about his bed. The diagnosis in this case was alcoholic paralysis, with dilatation of the heart, and the patient was ordered a diuretic mixture with tincture of digitalis.

August 13.—The patellar tendon re-actions are found absent, but the plantar reflexes are still active.

August 31.—Since his admission to the Infirmary the patient has become gradually weaker. Both hands are now manifestly dropped at the wrist, special movements of the fingers are imperfectly performed, and the muscles of the thenar and hypothenar eminences are specially atrophied, while there is a diffused wasting of the muscles of the upper extremities generally. As the patient lies on the bed, with the legs extended, both feet are dropped at the ankles. The power of producing dorsal flexion of the feet is much impaired, and during the attempt to effect this movement the feet become inverted, owing to the predominant action of the tibialis muscles, the tendons of which may be seen to become prominent in front and above the ankles. He can still raise each leg from the bed in an extended position, and can flex and extend at the knees, but all the movements are feeble. The muscles of the lower extremities generally have undergone a diffused wasting.

The patient states that his hands and feet feel numb, and the tactile sensibility is much impaired in the skin of the hands, feet, and legs. He still suffers from occasional attacks of cramp of the calf, and the muscles are very tender to pressure. The voice has for some time been feeble and husky, and now there is almost complete aphonia. The knee jerks, the cremasteric, the abdominal, and the epigastric reflexes are absent, but the plantar reflexes still persist. For the last two weeks there has been dribbling of the urine, as well as involuntary discharge of fæces. The pupils are equal, and re-act to both light and accommodation. The optic discs are found normal on ophthalmoscopic examination. The deafness persists. A tuning fork is heard at three inches on the right, and at eight inches on the left side, but when placed on the vertex it is heard better on the right than on the left side. Examination by the speculum shows old perforation of the right membrane which is covered by a thin cicatrix, strongly reflecting the light. Since the patient's admission the temperature has risen on one or two occasions to near 100° F., but on the whole it has been near the normal until two days ago, when it rose to 100·5° F., and now it stands at 102° F. The electrical re-actions of the nerves and muscles were examined to-day. All the muscles and nerves give re-actions to weak faradic currents.

Galvanic reactions of muscles:—

	LEFT ARM.		RIGHT ARM.	
	KSC.	ASC.	KSC.	ASC.
Extensor communis digitorum. No. Cells Leclanché	20	20	20	20
Extensor ulnaris	20	25	20	25
Supinator longus	20	25	25	25
Flexor carpi radialis	25	30	25	25
Flexor ulnaris	20	25	25	25
Flexor sublimis	25	25	25	25
First interosseous	25	25	30	25
Second interosseous	20	20	25	25
Third interosseous	25	30	25	25
Fourth interosseous	25	30	30	25
Flexor brevis pollicis	25	25	—	—
Adductor pollicis	—	—	35	25

	LEFT LEG.		RIGHT LEG.	
	KSC.	ASC.	KSC.	ASC.
Extensor longus digitorum	20	25	25	30
Tibialis anticus	20	20	15	20
Peronei	25	25	30	35

The general result of the galvanic examination of the muscles is that anodal closing contraction is greater than cathodal closing contraction in the adductor pollicis and interosseous muscles of the right hand and the second interosseous muscles of the left hand, while the two are equal in the extensor communis digitorum of the left arm. These muscles then alone show the first indications of the reversal of the normal formula which constitutes the re-action of degeneration in its partial form.

For the last two days the patient's breathing has become somewhat accelerated, and to-day the respirations number thirty-nine in the minute. The breathing is now of the upper costal type, and at each inspiration the epigastrium falls in, while the ribs seem to become very expanded and prominent at their lower margins and form a strong contrast with the sinking in of the abdomen and epigastrium. The patient is quite unable to strain, and during his efforts the epigastrium does not become protruded. The abdominal muscles fail to become tense when the patient makes an effort to raise his head and shoulders. His attempts at coughing are short and ineffective, and the displacement of muscles which it causes seems not to pass beyond the trachea at a point just above the supra sternal notch. The patient is apathetic, but his countenance is calm, and although the breathing is accelerated there are no paroxysms of dyspnœa and no great lividity of the face. The difficulty of breathing increased during the day, and the patient died from asphyxia on the following morning.

Permission for a complete autopsy was refused, but Dr. Williamson secured some of the peripheral nerves, and the following is his report of the changes found in them on microscopical examination:—The trunks of the musculo-spiral, posterior interosseous, external popliteal, and anterior tibial nerves, with their smaller branches, were examined. To the naked eye the nerves appeared normal. They were hardened in Müller's fluid, embedded in celloidin, and cut in transverse and longitudinal section. Some sections were stained according to Weigert's method, others with logwood. Pieces of the smallest branches were teased out. Sections of all the preparations were stained by Weigert's method, but the smallest fibres were also stained with alum-carmine, and osmic acid.

The most marked changes were found in the smaller branches of the nerves, the changes being most distinct in the finest branches and least marked in the largest nerve trunks. Microscopical examination showed extensive degeneration of the nerve fibres, a few healthy ones being found alone in some of the nerve bundles. In the smaller bundles the fibres were widely separated from each other, instead of lying close together as in health. The intervening tissue was formed of connective tissue, in which was embedded a large number of round or oval nuclei, while it presented a large number of small, more or less round, spaces, representing the position of the nerve fibres which had disappeared.

In Fig. 1 the nerve fibres are seen to be widely separated from one another by a granular-looking tissue, in which many round or oval nuclei are seen.

In Fig. 2 the black rings and black dots represent the nerve fibres which have persisted, and these, instead of lying close together as in health, are widely separated by a light brown tissue of granular appearance, and containing many small, more or less circular, clear spaces, that represent the position of the atrophied nerve fibres.

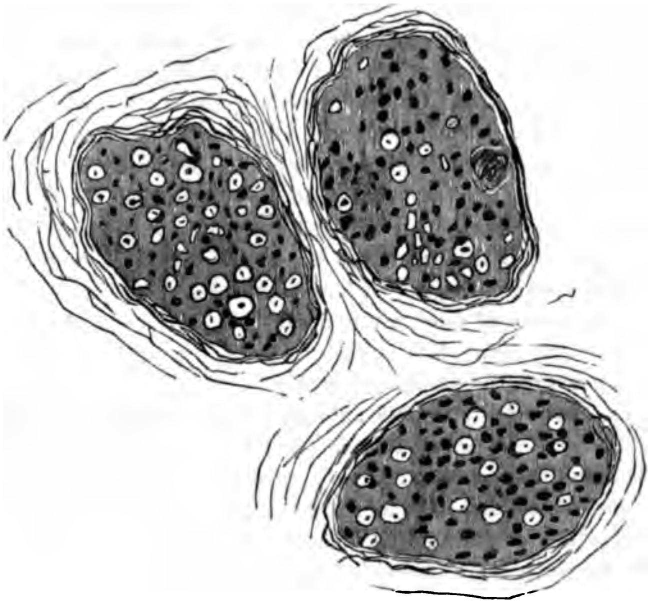

FIG. 1.—Transverse section of small branch of anterior tibial nerve, from a case of alcoholic paralysis (Case 6), stained by logwood.

A small branch of the posterior interosseous nerve was teased out, and stained by Weigert's method. The white substance of Schwann was seen to be broken up into more or less globular masses of variable size, which at certain points were collected into clusters, so as to cause the fibre to appear swollen, while at other points both the white substance and axis cylinder had completely disappeared.

The notes on the following case were taken by my clinical clerk, Mr. H. Part:—

Case 7.—R. B., aged 35 years, was admitted on December 30th, 1889, to the Manchester Royal Infirmary, under the care of Dr. Ross. The patient states that he is an artist by occupation, and appears to have enjoyed good health until some months ago, when he began to drink very heavily. For the last three months he has taken very little food, but has drunk large quantities of beer. About a month ago he began to suffer from severe cramps of the calves of the legs, which usually attacked him soon after he got into bed. At this time he felt his feet and hands numb and dead, and was also subject to severe shooting pains in the lower extremities. Soon afterwards he experienced difficulty in walking, and the feebleness of the lower extremities increased so rapidly that in a few days he was unable to stand.

Present Condition.—The patient lies on his back in bed, and is unable to move the lower extremities. Both feet are dropped at the ankles and inverted, and all the toes are hyper-extended at the metatarso-phalangeal and flexed at the phalangeal joints. The patient can perform all the movements of the upper extremities, including the special movement of the fingers. The abdominal wall is sunk in, especially at the epigastrium, and the lower costal borders are very prominent, while the normal rhythm of the respiratory movements is altered in such a way that the epigastrium falls slightly inwards during inspiration, and is slightly protruded during expiration. The patient's cough is so feeble and ineffective that the accumulated mucus seems never to be displaced by it beyond the trachea, while the epigastrium is not in the slightest degree protruded during the expiratory effort. The patient is quite unable to raise himself in bed, and when he attempts to do so the abdominal muscles remain quite flaccid under the observer's hand. There is also no protrusion of the abdomen during attempts at straining. The muscles innervated by the cranial motor nerves are free from manifest paralysis. The patient complains of numbness and tingling of the hands and feet, and of a peculiar sensation in the

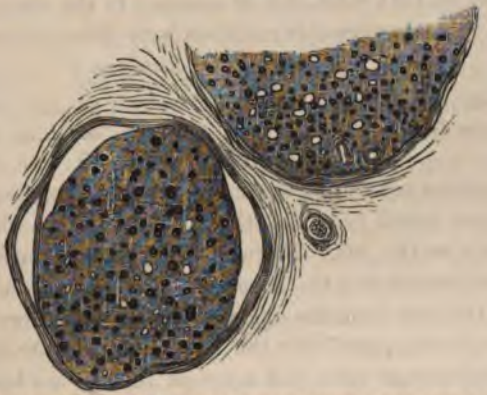

FIG. 2.—Transverse section of small branch of musculo-spiral nerve, from a case of alcoholic paralysis (Case 6), stained according to Weigert's method.

abdomen, which he says "feels as if it did not belong to him." There are, however, no manifest sensory disorders to objective examination. He can distinguish, with closed eyes, between the head and point of a pin over both legs, and can also localise correctly the point touched by the head of the pin.

Both knee jerks are absent. The plantar reflex is lost in the right foot, but is present in the left. The cremasteric and abdominal reflexes are feeble, but not quite lost. The patient experiences some difficulty in voiding his urine, but this appears to be due to his inability to strain, owing to paralysis of the diaphragm and abdominal muscles. The rectum is unaffected. The tibiales antici muscles and the extensor longus digitorum of the left leg re-act to a faradic current of medium strength, but the extensor longus digitorum of the left leg, and the peronei muscles on both sides, fail to contract to the strongest current. The following galvanic muscular reactions were obtained :—

		RIGHT LEG.		LEFT LEG.	
		K.S.C.	A.S.C.	K.S.C.	A.S.C.
Tibialis anticus	Cells Leclanché	30	30	30	30
Extensor longus digitorum	„ „	30	35	30	35
Peronei	„ „	35	35	35	40

The respirations are 28 in the minute, and the breathing is laboured and of the upper costal type, but the right side of the chest does not expand so freely as the left. There is great dulness in front over the right apex, and behind over the superior scapular region, and again over the whole of the base from the angle of the scapula downwards. The vocal fremitus is diminished over the right base, and the breath sounds are feeble. Submucous râles, and sonorous rhonchi are heard over the whole of the right lung, and fine crepitations are heard over the base on the same side. The temperature is 99° F. The pulse is feeble, and beats 132 in the minute. The optic discs are of a greyish red colour, but the margins are fairly distinct; there is no swelling, and the vessels are not tortuous. The urine is acid, and the specific gravity is 1020; it deposits urates on cooling, and contains a trace of albumen. The tongue is coated by a thick fur, which is white and moist at the sides, but of a dark brown colour, and dry in the centre.

December 31.—The temperature rose to 101·2° F. last evening, and now stands at 100·5° F. The respirations are 32 and the pulse 138 in the minute. The paralytic symptoms are unchanged, except that the big toes, which were hyper-extended at the metatarso-phalangeal joints yesterday, are now dropped at both joints.

January 9.—For the first six days after admission the temperature has been of a remittent type, varying from 99° F. to 100° F. in the morning and from 100·5° F. to 101·5° F. in the evening. For the last two days the temperature has gradually risen until it attained 104° F.

last evening, and now stands at 101·8° F. The respirations have varied from 28 in the minute, the lowest, to 44, the highest; and the pulse from 130, the lowest, to 160, the highest. The patient is becoming manifestly weaker, but there is no marked change in the paralytic symptoms.

January 14.—During the last three days the upper extremities have been the subjects of marked tremors, while there have been incessant twitchings of the fingers and face. The urine has been passed unconsciously in bed, and, from a state of low muttering delirium, the patient passed, yesterday, into a condition of complete unconsciousness. He died during the day.

Post-mortem examination.—The autopsy was conducted by Dr. Wild, pathologist to the Infirmary, thirty hours after death. The body is that of a spare adult male. Rigor mortis is present in the face and extremities. The pupils and orifices are normal. The feet are seen to be in dropped positions. The pleural and pericardial cavities contain a small quantity of fluid. The heart weighs 10 ounces. The valves are competent—the tricuspid orifice admits three and the mitral two fingers. The internal surface of the aorta is covered by small patches of atheroma, and patches of thickening are found at the bases of the tricuspid and mitral valves. Extensive pleuritic adhesions are found at the apex of the right lung, and the right lung itself is seen to be profoundly affected by tubercle, the lesion being more marked and of older date in the superior than in the other lobes. The superior and inferior lobes present a large number of scattered patches of caseous pneumonia. In the lower lobe the patches are discrete, and vary from the size of a pin's head to that of a pea, the smaller of the two being probably miliary tubercles. The patches are often so closely aggregated that only small tracts of air-containing pulmonary tissue intervene, and these are in a state of congestion. In the superior lobe the patches are less numerous, but each is of larger size than those found in the inferior lobe. The apex is occupied by extensive tracts of homogeneous caseous material, whilst in the middle of the upper lobe two cavities—one about the size of a walnut and the other considerably smaller—are found, having irregular and ragged walls crossed by fibrous bands.

The left lung is free from adhesions, but the upper lobe contains a few small consolidated lobules, whilst the whole lung is greatly congested, although still crepitant. The liver weighs 54 ounces. Its surface presents a few scattered raised patches, and, on section, each of the lobules are seen to be pale at their centre and congested at the periphery. The spleen weighs 6½ ounces, and is soft and congested. The kidneys weigh each 7 ounces. The capsule strips off readily, the surface is covered by stellate veins, and the cortex and medulla are of normal size and appearance, but both are rather soft and dark in colour. On removing the calvaria, the membranes of the brain show some opacities, and there is an excess of fluid in the lateral ventricles. No lesion is discovered on slicing the brain.

The spinal cord, anterior tibial nerves, the vagi, and the phrenic nerves were retained for microscopical examination. The microscopical examination was conducted by Dr. R. T. Williamson, to whom I am indebted for the following report:—

Portions of the anterior tibial nerves were teased out, stained in osmic acid, and mounted in glycerine, and on being examined it was found that the nerve fibres in the small branches had undergone marked changes. Similar changes were also noted in the nerve fibres of the main trunk, the alterations being more marked in the lower third than in the upper two-thirds of the nerves. In most of the nerve fibres the white substance of Schwann, which is stained of a black colour in the specimens (Fig. 1) is seen to be broken up into separate masses or segments. These segments form, at some points in the course of the fibres, oblong or oval swellings, and at other points large spherical masses which are made up of an aggregation of small globules. In the intervals between these segments, which in some places extend over a considerable distance, the fibre is destitute of medullary sheath, and looks small and wasted, while in other intervals even the axis cylinder has disappeared, and the fibres, being represented by the elastic sheath alone, look like a thin, transparent, and delicate filament.

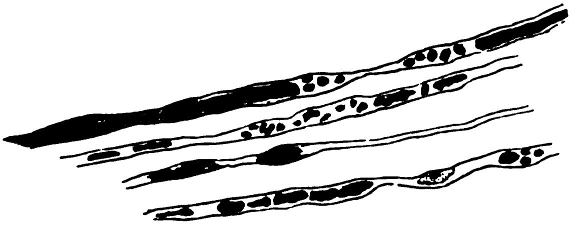

FIG. 1.—Fibres of the anterior tibial nerve stained by osmic acid.

Transverse sections of the anterior tibial nerves were stained by Weigert's method, by means of which the outer part of the white substance of Schwann is, in normal nerves, stained of a black, and the non-nervous elements of a brown colour. In these sections the nerve fibres which were stained of a black colour, and which may consequently be regarded as more or less normal, are separated more widely from one another than occurs in a healthy nerve by tissue stained of a brown colour. The increase of the brown stained tissue appears to be due partly to the disappearance of many of the nerve fibres and partly to the growth of new connective tissue between the fibres. In many of the transversely cut fibres the white substance appeared as an aggregation of globules instead of presenting the black rings which indicate normal fibres.

A portion of a small terminal muscular branch of one of the phrenic nerves was teased out and stained according to Weigert's method, and well marked changes were found in the nerve fibres (Fig. 2), these being in all respects similar to those already described as occurring in the fibres of the anterior tibial nerves. A small pulmonary branch of the vagus (Fig. 3), was treated in the same way as the filament of the phrenic, and its fibres were found to be altered in the manner already described as occurring in the fibres of the anterior tibial nerves.

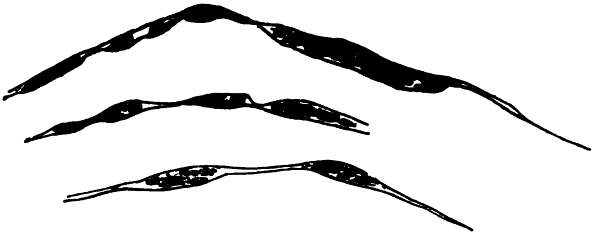

FIG. 2.—Fibres from terminal muscular branch of phrenic nerve, stained by Weigert's method.

A small portion of the extensor longus digitorum muscle was teased out and stained with logwood, and it was found that the transverse striation was quite as well defined as in healthy muscle fibres, but the muscle corpuscles appeared to be increased in number. A portion of the muscle of the heart was examined, but no manifest changes could be discovered in the fibres.

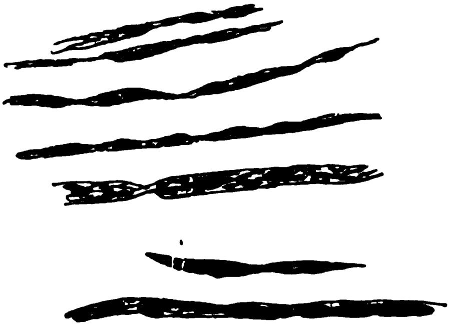

FIG. 3.—Fibres from small pulmonary branch of vagus, stained by Weigert's method.

Transverse sections of the spinal cord were stained by Weigert's method, and it was found that some of the ganglion cells of the anterior horns were probably somewhat destitute of processes, and altered in other minor respects, but the changes were exceedingly slight and doubtful. The vessels were not distended, and their walls did not appear to be thickened or otherwise altered. Deiter's cells were not increased in number, and it was particularly noted that the columns of Gall showed no trace of morbid change.

The following notes were taken by my clinical clerk, Mr. W. C. Wright :—

Case 8.—J. R., aged 45 years, was admitted to the Royal Infirmary, under Dr. Ross, on March 12th, 1890. The patient is a plumber by trade, and five years ago he suffered from an attack of lead colic, and at the same time he had a painful swelling of his feet, which was regarded as gout by his medical attendant. At Christmas last he suffered from an attack of influenza which confined him to bed for a fortnight, and from which he made but a slow and imperfect recovery. He admits that he was always a free beer drinker, but some months ago his wife died, and since that time, in order to overcome his feeling of dejection, he indulged to excess, drinking on an average as many as five pints of beer each day. Since his wife's death he has suffered from a diffused tenderness of the scalp, extending from the nape of the neck over the back and on to the top of the head. This headache was accompanied by sleeplessness and great mental depression. On closing his eyes for sleep he has seen images of persons floating before him, and at times they were so vivid that he believed them to be real, while the little sleep he obtained was much disturbed by horrid dreams. He also complained of pains along the spine. He had an attack of gonorrhœa when a young man, which was followed by buboes, but he never had syphilis. His wife never had miscarriages or dead-born children.

About a fortnight before his admission the patient was suddenly seized with giddiness, headache, and pains in the stomach, which were attended by copious and repeated vomiting, and he continued to suffer from these symptoms until his admission to the Infirmary. On the day after the vomiting commenced he felt great weakness in the lower extremities, and three days later the hands began to drop at the wrists. He now began to suffer from darting pains in various parts of the body, but which attained their maximum intensity in the shoulders and arms, the lower extremities, and the loins. On the day before his admission he noticed that the outside of his legs and the inner borders of the forearms and hands had become numb and without feeling. Five years ago he suffered from painful cramps in the calves of the legs, but they have not been troublesome during this illness. The patient states that he could walk a little up to the date of his admission, although he felt his legs very feeble and stiff.

Present Condition.—The patient has sandy whiskers, a fair skin, and florid complexion, but without having in any way a bloated appearance. A careful examination of his gums by the aid of a lens fails to reveal the slightest trace of a blue line. He lies on his back in bed with the lower extremities fully extended at all the joints, and their muscles are seen to be much wasted, the atrophy being very marked in the muscles of the calves and the anterior muscles of the legs. The upper part of the dorsum of each foot is on a plane continuous with the anterior surface of the leg, while the inferior half of the foot is drawn downwards towards the sole, so that the instep is strongly arched from above downwards with the convexity directed upwards. The heel is drawn upwards in a position of extreme dorsal extension, and the metatarsal bones are so strongly flexed towards the sole that the ball of the big toe is nearly on the same plane as the heel (Fig. 4). The toes are hyper-extended at the metatarso-phalangeal and flexed at the phalangeal joints. The patient can by voluntary effort produce slight flexion and extension of the toes, but he is quite unable to move either foot in the slightest degree at the ankle, and it is even found impossible to produce, except to a very slight degree, dorsal flexion of the foot by passive movements. The muscles of the calf and the tendo-achillis are found to be in a state of tension when the limb is at rest. This tension is much increased in degree at every attempt to produce dorsal flexion of either foot, so that it is quite manifest that the position occupied by the foot is less a result of paralysis of the anterior extensors of the foot than of persistent contraction of the muscles of the calf. Some degree of resistance is offered by the extensors of the thigh to flexion at the knee joint; but the patient can flex the leg on the thigh by voluntary effort, and he can also lift either lower extremity from the bed as a whole until the heel is raised from eight to ten inches from the plane of the bed. The patient can perform abduction and adduction of the thighs, and flexion and extension of the hip joints without difficulty.

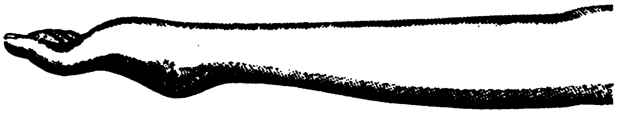

FIG. 4.

The patient is unable to stand without support, but if, when he sits on the edge of the bed, he gets his arms planted over the shoulders of an attendant placed on each side of him, he is able by a sudden effort to shoot suddenly into the erect posture, and he then stands on the extreme ends of the balls of his toes, his heels being raised about four and a half inches from the ground. The effect of this sudden action is extremely comical. The patient, to begin with, is a tall man of about six feet, and when to this height another four and a half inches is added by the raising of the heels, he attains to a gigantic height. He has a long, lank face, with lantern jaws, and lugubrious expression, reminding one of Doré's illustration of the "Knight of the rueful countenance," and when he steps forwards slowly on tip-toes, his long, thin, spindle shanks, and stiff gait, bear a marked resemblance to the walk of a huge stork, a comparison aptly made by one of my clinical clerks. Of more scientific interest, however, is the fact that at each forward step the heel of the foot planted on the ground is subject to a succession of slight depressions and elevations, not unlike that which occurs in lateral sclerosis, and which is suggestive of being of the same nature as ankle clonus. No clonus, however, can be elicited at the ankle by the usual manipulations, and the patellar tendon reactions are absent. When the arms are held out horizontally there is marked double wrist drop. The patient's grasp is feeble, and he cannot, without great effort, oppose the tip of the thumb to the tips of the fingers. The extensor surface of each forearm is markedly wasted, the interosseous spaces are grooved on the backs of the hands, and the thenar and hypothenar eminences are flattened. The external and posterior surfaces of each leg and the dorsum of each foot is more or less anæsthetic, and over these parts the patient, with closed eyes, is unable to distinguish the head of a pin from its point, and he does not localise accurately the part touched. The skin over the little and ring fingers and the ulnar half of the hand, both back and front, are similarly affected by anæsthesia. The patient is also unable, with closed eyes, to tell the position in which his legs may be placed by passive movements. The muscular masses generally, and especially those of the calves of the legs, are very tender to pressure. The ocular and facial muscles are unaffected, but difficulty is experienced in protruding the tongue, and he can only project the tip a very slight distance in front of the teeth. It also seems to deviate slightly to the right on protrusion, and is very tremulous. The plantar reflex does not appear to be completely lost on either side : the cremaster reflex is very feeble, but the abdominal and epigastric reflexes are active.

The patellar tendon reactions are, as already mentioned, lost, and the wrist and triceps jerks are also absent. All the affected muscles, both in the lower and upper extremities, react to a weak faradic current. The following galvanic reactions were obtained :—

		RIGHT LEG.		LEFT LEG.	
		K.S.C.	A.S.C.	K.S.C.	A.S.C.
Tibialis anticus	Cells Leclanché	25	35	25	30
Extensor longus digitorum	„	30	40	25	30

The pupils are moderately dilated, equal, and react to light and accommodation. The optic discs are normal. The temperature was somewhat raised on his admission, fluctuating for the first three days from 99° F. in the morning, to 101·8° and 102·6° F. in the evening. On the fourth morning it fell below normal, and after fluctuating slightly about 99° F. for the next two days, it then fell to 98·5° F. and remained normal during his subsequent stay in the Infirmary.

For the first few days after his admission the patient suffered from griping pains in the stomach, nausea, and vomiting, while his bowels were constipated. The lungs are found to be normal on physical examination, the cardiac dulness is not enlarged, and the sounds are not accentuated or accompanied by murmur. The areas of hepatic and splenic dulness are normal. The urine is normal in quantity, it gives a neutral reaction, the specific gravity is 1024, and it contains a trace of albumen, but no sugar.

The occipital headache from which he suffered before his admission persisted for some days. On examination of the spine the spinous process of the fourth dorsal vertebra is tender to pressure, and a band of hyperæsthetic skin passes on this level round each side of of the body and is well marked just below the nipple in front. The spinous process of the first lumbar vertebra is likewise tender to pressure, and the area of skin supplied by the sensory branches of the first and second lumbar nerves is very sensitive to pinching, the skin supplied by the iliac and inguinal branches being particularly sensitive. The patient was ordered 5 grains of pot. iod. three times a day, and a draught of 20 grains each of potass. brom. and chloral hydrate at bed time.

April 1.—The patient has greatly improved in his general health since his admission. The occipital headache, and in large measure the depression of spirits and sleeplessness have disappeared, and he enjoys his food. On standing, the heels are now 2½ inches removed from the ground, but the spasm of the gastrocnemii still persists, and the paralysed muscles have not improved much in motor power. The patient was discharged at his own request.

Alcoholic paralysis comes so frequently under the notice of physicians in Manchester, both in hospital and in private practice, that it would not be difficult to multiply records of cases, but those just described will, I trust, suffice to give an accurate picture of the symptoms and course of the disease. In addition to the eight cases here reported and five published elsewhere,* I have collected 77 from medical literature, making in all 90 cases, and have analysed them in accordance with the tabular scheme adopted in dealing with Landry's paralysis. To reproduce these tables, however, in full would occupy too much space, and even to describe the results obtained by analysis of them in detail would be tedious, without carrying with it compensating advantages.

With regard to the cause of the disease it is manifest that no case has a right to be included in our list unless the symptoms are at least supposed to have been caused by alcoholic excesses. It is thought that in several of the recorded cases there was a possible complication of syphilis, in a few more the lungs were affected by tubercle, in several the appearance of albumen in the urine, taken with other symptoms, pointed to the presence of granular kidney, and in one case reported by myself the occupation of the patient as a painter rendered the presence of lead probable, although no blue line was found in the gums; but in all these cases the clinical picture of the disease, as well as the pronounced habits of the patients immediately before the onset of the symptoms, amply attested that alcohol was the active agent in the production of the paralysis. With regard to the form in which the alcohol is taken the information obtained in the records of cases is not so precise as to enable one to arrive at definite conclusions. In 22 cases the patients are said to have drank large quantities of spirits in the form of brandy, whiskey, gin, or rum; 3 took absinthe or vermouth; 13 drank beer and spirits; and 5 drank beer only. Of the remaining 47 cases it is stated generally of 30 patients that they "drank to excess," or were "heavy drinkers," and of the other 17 patients excessive indulgence in alcohol is inferred from such facts as that the patient was known to have suffered from an attack of delirium tremens, or from morning vomiting, and other characteristic symptoms of this form of poisoning.

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* Ross (J.). "The premonitory symptoms of alcoholic paralysis."—*The Lancet*, Vol. I., 1889, p. 1125.

It has often been stated that alcoholic paralysis is always the result of spirit drinking, but it will be seen that in five out of the 90 cases here collected the patients drank beer only. Judging, however, from my own experience, I am of opinion that beer drinking is a much more frequent cause of alcoholic paralysis than this estimate of less than 5 per cent of all cases would represent, and I am glad to be able to say that my friend Dr. Reynolds has, from careful statistical evidence, which I trust he will publish soon, come to the conclusion that, in our Infirmary patients, both alcoholic paralysis and cirrhosis of the liver are caused more frequently probably by beer drinking than by any other form of alcoholic indulgence.

Another statement frequently made is that alcoholic paralysis generally attacks the female sex, but of the 90 cases here collected, 41 only were females. It does not even appear from these cases that the disease is much more fatal in women than in men, inasmuch as out of the 49 males attacked, the disease proved fatal in 14 cases, and of the 41 females, it was fatal in 13 cases. At the same time it must be admitted that the worst cases of paralysis—those in which the hands and feet become dropped at the wrists and ankles, in which the muscular masses of the extremities undergo so much wasting that the bones seem to underlie the skin, and in which the paralysis proves altogether intractable to treatment—are much more frequently met with in women than in men, and although the absolute number of women attacked by paralysis may not exceed the absolute number of men, yet if the large number of the male community who habitually drink to excess be compared with the relatively small number of females who give way to excessive indulgence, it will be found that a much greater proportion of the latter are attacked by paralysis than of the former. The reasons for the preference with which women are attacked are not far to seek. Something is doubtless due to original delicacy of constitution, but much more to the fact that women, from having to attend only to household duties, are enabled to indulge at all hours of the day, and thus become more or less continuous drinkers, while men, from having to attend to definite occupations, often under masters, are obliged to have their drink in a more or less intermittent manner, and are often compelled to go many days at a time without any. When a man suffers from a minor degree of paralysis he is obliged to give up work, and his income and consequent power of access to his usual beverage generally becomes restricted, but a woman can lie in bed the greater part of the day and go on with her indulgence long after the paralytic symptoms have declared themselves. The very magnitude of the disgrace which attaches to drunkenness in women has often an unfavourable influence upon her, inasmuch as it drives her to secret drinking, which has a much more debasing effect upon her moral nature than if the sentiments of the community had enabled her to drink openly as men do. And when once the moral nature is undermined, all self-control and self-respect are lost, and the woman gives way unrestrainedly to her indulgence, and thus enters upon the downward path from which there is no recovery except by means of extraneous help. A man often stops voluntarily in his downward progress, even after a long career of dissipation—a woman hardly ever, and if she is rescued it must be by the compulsory efforts of her friends. It is also possible that the sedentary life which women lead is favourable to the deleterious action of alcohol upon the tissues; for it is a common experience that persons can drink a greater quantity of spirits when walking in a mountainous district like Scotland without feeling any of its immediate ill effects than when leading sedentary lives in their offices and counting-houses. Strong and well-nourished muscles involve a corresponding development of the peripheral nerves, and well-nourished nerves mean, other things being equal, greater resisting power against deleterious influences in general, alcohol included.

With regard to age, of the 90 cases collected, 13 occurred between 20 and 30, 29 between 30 and 40, 35 between 40 and 50, 10 between 50 and 60, and one only above 60 years, while in two the age is not stated. It will thus be seen that by far the majority of the cases occur between 30 and 50 years of age, and that the affection is seldom met with under

20 or over 60 years. Nothing very definite can be made out with regard to the relation of the disease to occupation, except that it is frequently met with in butchers, commercial travellers, and, above all, in beerhouse keepers and publicans. Of the women a few are said to have been prostitutes, while one or two are said to have been landladies of public-houses, and these last have contributed a considerable proportion of the cases of the disease in women which have come under my own observation. Respecting the influence of heredity, the information obtained is somewhat scanty, although it is sometimes stated that the patient's parents, brothers, or sisters have been drinkers, and at other times a neurotic family history is mentioned. The above seems all that it is necessary to say with regard to the causation of the disease, and we must now proceed to consider the symptoms.

The *symptoms* of alcoholic paralysis need not detain us long, inasmuch as they hardly differ in any respect from the symptoms of Landry's paralysis, when the affection pursues an acute and fatal course, as in the case of R. B. (Case 7) reported here, or from the symptoms already described of idiopathic multiple neuritis, when it pursues a sub-acute or chronic course. The symptoms are usually ushered in by numbness and tingling of the fingers and toes, and shooting pains in the extremities, or occasionally encircling the body. Cutaneous hyperæsthesia is occasionally mentioned, but this symptom is always subordinate to the excessive tenderness of the muscles on voluntary exertion or the slightest pressure, and although this tenderness is not peculiar to it, perhaps in no form of neuritis is it so excessive as in the alcoholic variety. The accessible nerve trunks, such as the popliteal, musculo-spiral, and ulnar, have been found swollen and tender to pressure. In a large number of cases anæsthesia of the lower extremities, as far as the knees, and of the upper as far as the elbows, has been noted. The anæsthesia is, however, rarely complete. In some cases it appears as a loss of sensibility to painful impressions, or simply as delayed sensibility to pain, or in the form of double sensations, the prick of a pin being felt at first as a touch and then as pain. Tactile sensibility, generally maintained, has been found in a few cases to have been blunted, and the sense of temperature has been noticed to have been modified in such a way that all objects felt cold, or a cold body has been correctly appreciated, while a hot body gave to the patient the sensation of an electric shock. Persistent and aggravated attacks of visceral neuralgia are often present. Attacks of colic, not unlike that of lead colic, have been observed by my colleague, Dr. Dreschfeld, and I have seen several examples of this kind myself. There are some grounds for believing that females sometimes pretend to have a seizure of colic in order to excite sympathy, and have brandy prescribed as a remedy; but in one case that came under my observation the attack seemed terrible in its severity and quite destitute of any appearance of simulation. Pains in the stomach or attacks of gastralgia are also common in alcoholic subjects, and, indeed, the gnawing pain at the pit of the stomach, which is only allayed by taking spirits, constitutes one of the great difficulties in overcoming the habit. In a large number of cases the sensory affections consist of the paræsthesia already described, and no disorder is discovered on objective examination. In a case recorded by Dr. Bramwell, sensory disorders were absent at first, and only appeared in the form of marked "cutaneous" hyperæsthesia during the stage of convalescence.

The *motor* disorder begins occasionally abruptly and extends rapidly, but, as a rule, the onset is slow, gradual, and insidious, and it is then found that the patient has had, probably for months, difficulty in executing certain actions, like buttoning his clothes, and that he has at the same time lost all spring in his walk, and has experienced great difficulty in ascending a stair, while his gait has been unsteady. But symptoms of motor irritation are present long before there is any sign of paralysis. I do not allude to the fact that the patients often describe the paralysed limbs as feeling stiff, but to the presence of active spasms. These appear often in the form of tremors and twitchings of the extremities, but much more frequently as active cramps, which may attack the muscles of the forearms and hands, but are most persistent and troublesome in the muscles of the calves of the legs. These cramps are most troublesome soon after the patient goes to bed and just as he is about to fall asleep, or again on awakening in the morning. The affected calf becomes drawn up into a "lump," and the patient has generally to jump out of bed and press his foot on the floor. He has to rub the affected extremity with his hands, and from twenty minutes to half an hour may elapse before the spasm relaxes sufficiently to enable him to return to bed. Some of these patients find that standing on the cold hearthstone tends to relieve their sufferings, while others find greater ease from warmth, and these may have to seek the warmth of the fire before the spasm relaxes. The fingers also are liable to be attacked, and these spasms are particularly apt to come on while the patient is engaged in some kind of manipulation, such as sewing, writing, or playing the piano. These cramps have been mentioned by Hüss, Glynn, Taylor, and Oettinger, but it is curious to notice how little attention they have attracted considering that they are seldom altogether absent, and that in the majority of cases they distress the patient for years before the paralysis declares itself.

Contracture of the limbs, from adapted shortening of the comparatively healthy and unantagonised muscles, is not uncommonly met with in alcoholic paralysis, and the deformities thus resulting are rendered more persistent by the formation of adhesions in and about joints. The persistent contracture, however, observed in the last case (*Case 8*) here reported is comparatively rare, although more or less similar conditions have been reported by Oettinger, Taylor, and Saundby. The two first authors were of opinion that the persistence of the deformities in their cases was due to peri-articular adhesions, but in my case the vicious attitudes were clearly due to spasm of the muscles of the calves, inasmuch as a kind of clonus was induced in the foot which had to bear the weight of the body in walking. Dr. Saundby appears also to have been of opinion that the contracture of the knees and ankles in his case was due to muscular spasm, as he speaks of the presence of atrophic and spastic paralysis in discussing the symptom.

When the paralysis is fully constituted, the characteristic distortion of double ankle drop, followed later by double wrist drop, make their appearance, and the patient now manifests the well-known walk already described as the high-stepping gait. A careful examination, however, generally shows that the paralysis of the extensors, which causes the drop at the wrists and ankles, is preceded by various distortions of the fingers and toes, and inability to perform accurately and without tremor certain special movements with the fingers. Now, although the muscles which move the peripheral segments of the extremities are usually attacked in preference to the muscles attached to the trunk, this rule is not without important exceptions. I have known the symptoms to begin suddenly by shooting pains about the shoulders, and the deltoids were implicated in the paralysis at an early period of the disease. In the case of R. B. (*Case 7*) it was found that the diaphragm and abdominal muscles were completely paralysed on the patient's admission to the Infirmary, while the great acceleration of the pulse, which varied from 130 to 160 beats in the minute, indicated that the inhibitory fibres of the vagus were implicated.

It is true that the lower extremities were likewise completely paralysed, but the distortion of the feet and toes present on his admission only indicated the second degree of paralysis already described; while the hands, although feeble, did not show any distortion or failure in performing special movements. In this case, then, the splanchnic nerves were attacked in equal proportion with, or even in preference to, the somatic nerves.

The paralysis may extend to the muscles of the back and neck, so that the patient is unable to sit up in bed or to move his head on the pillow. The facial muscles are not, however, the subjects of decided paralysis. A certain degree of feebleness of them is usually present, as indicated by loss of expression, widening of the palpebral fissures, and tremors of the lips in speaking or on protrusion of the tongue. The tongue also is tremulous on protrusion—though not paralysed—and the patient's articulation is often so tremulous and indistinct that the disability may be readily mistaken for the dysarthria of general paralysis of the insane. The voice is sometimes feeble and husky, and at times there has been complete aphonia, and Bernhardt found in one case paralysis of the left vocal cord, and Taylor and Broadbent met with paralysis of both vocal cords. Dysphagia has been noted by Broadbent and Findlay. The ocular muscles are occasionally implicated in the paralysis. Nystagmus has been observed by Hadden, Oppenheim, Vierordt, Thomsen, and myself, and the oscillatory movements are often associated with diplopia and strabismus, double convergent squint from paralysis of the sixth pair of nerves being most frequently noted. Three cases of nystagmus in alcoholic paralysis came under my own observation, but in none of them could strabismus be discovered. In one of Thomsen's cases the eyes were directed to the ground, and the patient could not raise them by voluntary effort or direct them upwards and outwards; ptosis came on at a later stage, and the disease proved fatal soon afterwards. Wernicke appears to have been the first to have directed attention to the appearance of double ptosis in chronic alcoholism, three cases of which he mentions, all of which proved rapidly fatal. At the autopsy the nuclei of origin of the ocular motor nerves in the floor of the aqueduct of Sylvius, and the upper part of the fourth ventrical were studded with capillary hæmorrhages, and a similar pathological condition was found by Thomsen in two fatal cases which he reports. Wernicke proposed to name this affection *poliencephalitis superior acuta*, but the name does not appear to be well chosen, and in the meantime we prefer to regard it as one of the complications, or rather the extensions, of alcoholic paralysis. Two cases of the kind came under my own observation, and both died about 48 hours after my visit, but I had no opportunity of making an inspection. In some of the cases in which the external muscles of the eyeballs were paralysed, the pupils were in a medium state of dilatation, and reacted sluggishly to light. In a case reported by Oppenheim, mydriasis of both pupils was present, and the left pupil failed to react to the stimulus of light, while the right was motionless both to light and accommodation. The pupils have sometimes been found unequal. In one of Fischer's cases the pupils were contracted and fixed.

The *sphincters* are, as a rule, unaffected. Of the 90 cases collected, there was involuntary passage of urine and stools in four cases; of urine alone in four cases; retention of urine and stools in two cases; difficulty of micturition in one case, and the patient had to obey the call quickly, in order to avoid dribbling, in another case. The patient is said to have been impotent in four cases, and it is probable that strict enquiries would have revealed the fact that this function is often in abeyance.

The *reflex actions* are variously affected in alcoholic paralysis. Of all the reflexes the condition of the patellar tendon reaction is by far the most valuable sign of the disease. Out of the 90 cases collected this reaction is not mentioned in 13 cases. In 5 cases it is said to have been exaggerated or lively, in 1 case it was normal, and in another it was exaggerated at first, and became lost with the progress of the disease. It was absent in 5 cases, but returned as the patients improved, and it was completely lost in the remaining 65 cases. It is, indeed, of the utmost importance to remember that the knee-jerks are exaggerated in the early stage of the disease, inasmuch as an early recognition of the minor symptoms of this affection is of so much consequence to its treatment. In most of the advanced cases which become inmates of hospital wards these reactions are doubtless lost, but judging from my own experience they are decidedly exaggerated in the majority of cases which seek advice in private consulting rooms, provided the minor phenomena of the disease are searched for and recognised. The condition of the plantar reflex is not mentioned in 45 cases; it is said to have been normal in 7, feeble or retarded in 11, excessive in 5, and absent in the remaining 22 cases. The cremasteric reflex is said to have been normal or present in 6, excessive in 9, feeble in 6, absent in 6, and is not mentioned in the remaining 63 cases. The abdominal and epigastric reflexes were found normal or present in 9, active in 4, absent in 12, feeble in 5, and are not mentioned in the remaining 60 cases. Ankle clonus is mentioned as having been absent in a few cases, but as this reaction is not easy to evoke in healthy people its absence is not of much consequence. The wrist-jerks are also mentioned in a few cases as absent, and in one case in which the patellar tendon reactions were lost the triceps reaction at the elbow is said to have been retained. The mechanical contractility of the quadriceps femoris muscle is mentioned as having been lost in one case, and to be in excess in one case, and to have been in excess at first and then lost in another case.

The electrical reactions have been found somewhat variable, but the rule is for some form of the reaction of degeneration to be met with in the nerves and muscles most profoundly affected by paralysis. These reactions were not examined in 33 out of the 90 cases here collected. Of the 57 cases in which an electrical examination was made, it is stated in three cases that the faradic contractility was lost in the nerves and muscles most affected, and in eight cases that the electrical reactions were diminished. In 17 cases the faradic contractility was diminished in the nerves and muscles most affected, while the galvanic reactions manifested qualitative changes, constituting the partial reaction of degeneration, and in 29 cases the faradic irritability was lost in the nerves and muscles most affected, while the galvanic reactions of the muscles manifested qualitative changes, constituting the complete reaction of degeneration. The application of the faradic current to the phrenic nerves in the neck is said to have failed to contract the diaphragm in one case. On a survey of the whole evidence, I am inclined to think that although an electrical examination may be of use in forecasting the progress of a more or less chronic case towards complete or partial recovery, it is not of much value in judging of the gravity of the symptoms during the early stages of the disease. In the case of R. B. (*Case 7*), for example, the reaction of degeneration was noted in the anterior muscles of the legs only, and yet the diaphragm was already paralysed, and the patient died a fortnight after his admission to the Infirmary. In the minor degrees of paralysis, when it is of so much consequence that the nature of the disease should be recognised, the faradic and galvanic irritability are either normal, or only present such slight variations of excess or diminution in the reactions, that an electrical examination is of little or no value.

The *vaso-motor* symptoms consist of pallor of the hands and feet, caused by vaso-motor spasm, or redness and lividity of them which becomes especially marked when the limbs are dependent. The colour of the hands often changes rapidly from pallor to redness, according as they are held in the vertical or dependent positions. Closely connected with this vaso-motor mobility are puffiness and swelling, which are frequently observed on the backs of the hands and feet. One of the most characteristic symptoms of the disease is caused by vaso-motor spasm, which gives rise to great pallor, coldness, and "deadness" of the fingers and toes, or at times lividity of them—the condition which Raynaud has called "local asphyxia." In some cases this vaso-motor spasm proceeds to such a degree as to cause gangrene of the tips of the fingers. Attention was first directed by Lancereaux to the action of alcohol as a cause of symmetrical gangrene. A case of this kind came recently under my own observation. The patient was suffering from numbness and tingling of the extremities, nocturnal cramps, loss of the patellar tendon reactions, tottering gait, and other symptoms of alcoholism, and he made no secret of the fact that he was a great drinker. He had suffered for some time from alternating attacks of severe burning sensations in his hands and fingers, and coldness and deadness of them, and when I saw him the palmar surfaces of the two distal phalanges of the index and middle fingers of the right hand and the dorsal surfaces of the last phalanges were of a dark livid colour. The tip of the thumb was also similarly affected. Dark coloured bullæ had already appeared on the palmar surfaces of the affected digits, and the matrix of the nails had begun to ulcerate. In the early stage of the disease the soles of the feet and occasionally the palms of the hands are hot and burning, and often covered with a profuse and ill smelling sweat. The plantar nerves are likewise very sensitive to pressure, especially over the points where they bifurcate to form the digital branches, the condition being, indeed, very like the affection named by Dr. Weir Mitchell *erythromelagia.*

The *secretory* phenomena consist of profuse sweating, which is sometimes general, and at other times more or less restricted to certain localities, such as the forehead, the backs of the hands, and the feet. At an advanced stage of the disease the skin, especially that over the lower extremities, becomes excessively dry and covered by scales of dried epidermis.

The *trophic* phenomena consist chiefly of rapid emaciation of the muscular masses of the body and certain changes in the skin and its appendages. The affected muscles are, with the exceptions already noticed in which contraction was present, flaccid, and no tension is provoked in them by passive movements. They undergo rapid wasting, although the emaciation may be masked for some time by the presence of an excess of subcutaneous fat. But when the disease becomes chronic, the fat disappears, and the muscular masses are then seen to be greatly emaciated, the limbs being so thin and attenuated as to appear to consist of nothing but skin and bone. The skin loses its wrinkles and becomes smooth, that covering the fingers sometimes becoming hairless and polished, just like the "glossy" skin described by Paget as occurring in local injuries of nerves. A similar glossy condition is also sometimes seen in the soles of the feet. The nails sometimes become elongated and curved, and fissured longitudinally, and dry and cracked at their free edges, while the hairs are sometimes dry and brittle. In a case reported by Oettinger, and in another by Taylor, a bed sore appeared on the sacrum; and in one reported by Hadden, the legs were covered by an eczematous eruption and bullæ appeared on the right buttock and on several fingers of the right hand. Such occurrences are, however, rare in this affection. In a case reported by Schulz some of the joints were swollen and red, and I have myself met with two cases recently in which many of the large and small joints were inflamed, and in which alcoholic excesses had preceded the onset of the paralysis, but in such cases there is always a possibility of a rheumatic complication to account for the arthritic affection.

The hæmoglobin seems at times to undergo great destruction, so that a high degree of anæmia is often present, as in a case reported by Vierordt. A case of this kind came under my own observation. The patient, a lady, who had been occasionally under my observation for a considerable time, was dying from paralysis of the diaphragm when I last saw her. The pallor of the countenance, hands, and body generally, as well as of the lips and conjunctivæ, was extreme, and such as is seldom met with except in aggravated cases of pernicious anæmia, or in the cachexia of cancer. Connected with this blood change are most likely the attacks of epistaxis and hæmatemesis, which are occasionally mentioned amongst the symptoms. Hæmoptysis is also mentioned, but it probably is the result of pulmonary tubercle, which is frequently met with in alcoholic subjects. Purpuric spots are reported in two or three cases, and it is not uncommon to find in chronic drunkards the surface of the body covered with thousands of small purpuric spots about the size of a pin's head.

The *visceral* disorders and complications are numerous and important. The most common symptom of this kind is the morning retching, usually held to indicate the onset of cirrhosis of the liver, but in others severe vomiting, persisting for days together, is mentioned, and is most probably the result of the irritative action of strong spirits on the gastric mucous membrane. In 4 of the 90 cases exhausting diarrhœa is mentioned, and in one case the bowels are said to have been torpid.

The presence of albumen in the urine, either associated with hypertrophy of the heart, or with dilatation of the heart and general œdema, is mentioned in four cases. When cardiac dilatation and albuminous urine are associated it is difficult to determine whether the affection of the heart or of the kidneys has been the primary disease. It is very probable that in one class of cases the albumen is, like the general œdema, the result of venous congestion arising from tricuspid regurgitation; while in another class the cardiac dilatation is in great part caused by the high arterial tension which accompanies granular disease of the kidneys. This subject, however, is much too wide to be discussed at present. The pulse is generally feeble, irregular and frequent, the beats varying generally from 120 up to 140, or even 160 in the minute. In a case reported by Oppenheim, however, the pulse, which was irregular and intermittent, beat only 48 in the minute. The temperature is generally normal, except when there is a complication of bronchitis or tubercular phthisis, or when the breathing is embarrassed from paralysis of the diaphragm.

The *special senses* are not often affected in uncomplicated cases of alcoholic paralysis. In a case reported by Ballet, amblyopia was noted; but in this case, as in many similar cases, the patient was a heavy smoker, and it is consequently difficult to decide whether the visual defect is due to alcohol or to tobacco.

In one of the cases reported by Moeli pallor of the optic discs was found on ophthalmoscopic examination, and Strümpell found atrophy of the temporal half of each disc. A very important contribution to this subject has been made by Uhthoff. Out of 1,000 patients given to alcoholic excesses examined by him, he found in 139 the characteristic pallor of the temporal halves of the discs just mentioned. In 65 of those 139 patients no functional disorders of vision were detected, even although in 3 of these cases anatomical changes were found in the optic nerves after death. Slight disorders of function were, however, met with in 9 out of the 1,000 cases in which no abnormal changes were discovered in the discs on ophthalmoscopic examination. The author also found, in addition to the cases in which pallor was observed, slight cloudiness of the discs in 55 cases out of the 1,000, after excluding all cases which could have arisen from other causes than alcoholic poisoning. In three of these cases an autopsy was obtained, but a degenerative retrobulbar neuritis could only be proved in one of them. Of the cases in which pallor of the external halves of the discs were observed, 7 came to an autopsy, and 6 of these were solely due to alcoholic poisoning. In these 6 cases the author found an interstitial neuritis of the optic nerves, corresponding in extent to the pallor observed in the discs. It is worthy of note that the author has not been able to discover any sure signs by which to distinguish alcoholic and tobacco amblyopia from one another, and he comes to the conclusion that a particular form of retro-bulbar neuritis is the anatomical substratum of every form of toxic amblyopia.* The author found that 60 out of the 1,000 patients showed disorder of the pupil. In 25 cases the pupils were unequal, in another 25 cases the reaction to light was very slight, while in ten cases both the light reflex and the associated contraction to accommodation were absent. Paralysis of the ocular movements was comparatively rare. Nystagmus on lateral movements was present in 13 out of the 1,000 cases, and on voluntary movement in two cases, while pronounced paralysis of the ocular muscles was only met with in three cases, and in all of these the sixth pair of nerves was affected.

The *psychical* disorders of chronic alcoholism form a very important factor in the clinical picture of the disease, and they are exceedingly numerous as well as very varied in character. It is not our intention to describe minutely the mental disorders which attend the acute form of alcoholism or *delirium tremens*, or to discuss at length the various forms of insanity which result from long continued alcoholic excesses. It will suffice to mention briefly some of the more common mental aberrations met with in hospital and private practice, and the presence of which may be utilised in confirming our diagnosis of intemperance as the cause of the symptoms.

The psychical disorders of chronic alcoholism pass through a period of development or evolution, or it might be more correct to say that the patient's mind passes through various phases of disintegration or dis-solution, which, for the purposes of description, may be divided into four stages. It must not, however, be supposed that these stages are distinctly divided from one another, or that every patient passes successively through the symptoms which characterise each. These stages are: (1) A premonitory one in which the special senses and imaginative faculties are liable to be exalted; (2) a stage of depression or melancholia; (3) a transition stage of delirium, mania, or melancholia with excitement, or of convulsions passing on to (4) a final stage of dementia.

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* See Millingen (Dr. Van). "Toxic Amblyopia."—*Transactions of the Opthalmological Society*, Vol. VIII., 1888, p. 240, *et seq.*

The *stage of exaltation* may be illustrated by the psychical disorders observed in C. W. (Case 2). When the patient closed his eyes for sleep he saw a bright cloud, in the midst of which faces appeared which looked like photographs upon a coloured screen, but these hallucinations never imposed upon the patient as if they were realities. During his waking hours the patient seemed to have passed his time in a kind of fairy dreamland, but on being asked to describe his fancies he laughingly declined. Patients in this stage may fancy they hear some person singing in the distance, or a musical instrument being played in the next room, but the strain of music is hardly ever so vivid as to seem quite like reality. In moments of great excitement the patient's thoughts may be echoed in his ear, so that they seem to come from some external source. But with this exaltation of the senses the intellectual powers are always lowered, and the patient, while becoming more absorbed in contemplating his own thoughts and feelings, is becoming less observant of what is passing around him and less capable of discharging successfully the ordinary duties of life.

As the mental disorder advances he becomes less disposed to earn his living by steady industry, and is prone to endeavour to attain to riches, and not always unsuccessfully, by rash speculations and betting, and even in other less commendable ways. In this stage the thoughts are apt to become concentrated round one or two great schemes of ambition or revenge, and the subject becomes intolerant of everything that seems to him to throw an obstacle in the way of the realisation of his aspirations. A man in this state of mind will often bear with complete callousness the greatest calamities of life, such as the death of his wife or children, so long as these events do not seem to cross the line of his ambition; but the most trivial incident that appears to run counter to the realisation of his hopes evokes the most gloomy and painful emotions, and is apt to be met by a fearful outburst of mental irritability. Success in the line of his ambition carries with it greater danger to his mental balance than even the complete failure of his schemes. The effect of success in this stage is finely illustrated in Macbeth, a character which, if true to nature, must have been drawn from a subject whose turbulent and ambitious mind had been rendered still more unscrupulous by alcoholic excesses. Stripping from the drama the supernatural machinery of the witches employed by the poet, the facts are that Macbeth was returning home whilst labouring under the excitement caused by success in battle, when he is met by a messenger from the king who hails him as "Glamis, and Thane of Cawdor," and his own ambitious thought of "Hail, king that shall be" echoes in his ear as if it were uttered as a prediction from some external source, a prediction which he afterwards helped to realise by his active participation in the murder of Duncan. Viewed in this vulgar and prosaic form the character of Macbeth is one which is by no means uncommon in ordinary life, especially if we include cases in which the mental perturbation leads to the commission of other aggravated breaches of the moral law besides murder. The stage of exaltation sometimes culminates in an outburst of extravagant conduct, which may display itself in reckless speculations, or in the purchase of objects which are altogether beyond the means of the patient, and which are of no value to him when obtained. The extravagant acts and mental excitement and elation of this stage are, indeed, not unlike the early symptoms of general paralysis, and the similarity between the two affections becomes all the more striking when the alcoholic psychical disorder is conjoined, as frequently happens, with tremors of the tongue and lips, and unsteadiness of the hands in executing small movements, like buttoning the clothes or writing.

The *stage of melancholia* is characterised by great restlessness, moroseness, mental irritability, insomnia, and horrid dreams. It is also the period of self questioning, of soliloquy, of remorse, and occasionally also of repentance and reformation. Melancholia is mentioned as having been present in four only out of the ninety cases here collected, but it must be remembered that in a large proportion of the reported cases the patient had already attained to the stage of delirium, and the antecedent stage of melancholia may have been overlooked. This stage is often also completely masked by the large quantities of alcohol imbibed by the patient, and thus in many of these cases the stage of delirium follows so quickly upon that of exaltation that the period of depression is so inconspicuous and transitory as to be readily overlooked. The hallucinations of this stage are apt to become more vivid than in the first stage, although the patient is generally still able to correct the tendency to regard them as realities. The hallucinations are vivid but corrigible. Macbeth, who had manifested symptoms of the stage of melancholia before the murder of Duncan by his hesitation and soliloquies, was able at once to correct by a simple experiment, his first tendency to regard the phantom dagger as a real one, and on failing to grasp it he at once concluded that it was "a dagger of the mind, a false creation, proceeding from the heat-oppressed brain." In the case of a turbulent spirit like Macbeth, who had been hurried to commit a great crime, the period of melancholia passed quickly into that of mania, with its delusions of suspicion; but in other cases this stage may supervene slowly and gradually, and last for months or years before the stage of delirium and delusion is reached. In these chronic cases the stage of depression is ushered in by restlessness and sleeplessness. Insomnia is mentioned in eight out of the ninety cases here collected, but in three of these cases the sleeplessness was associated with rambling or other symptoms of delirium, so that five only of those cases can be regarded as belonging to this stage. The insomnia, however, of the stage of depression is a much more frequent symptom than those figures would lead one to suppose. Those who take alcoholic stimulants in what is usually regarded as moderate quantities have often a morbid dread of not sleeping on going to bed, long before they manifest any other sign of mental derangement, and if the physician protests against two glasses of whisky being taken at bedtime, the reply often is, "I cannot sleep without it." The dread of lying awake at night arises from the fact that already the disagreeable incidents of life with their associated painful ideas and gloomy thoughts are apt to obtrude themselves, and when a further stage of depression is reached the mind is apt to be excited at night by thoughts which chase one another in serial order with lightning speed, or to be agitated by a tumultuous tempest of conflicting thoughts and passions, which altogether prevent sleep unless the brain is drenched by alcohol or other narcotic. In other cases these symptoms of insomnia and restlessness do not rise for a long time to great intensity, and these, along with the gloomy thoughts and remorseful feelings are combatted and kept in check by the repeated doses of alcohol taken during the day, and the patient is thus able to attend in some sort of fashion to his business, although attention to the ordinary details of business is a source of great worry to him. Worry in business is doubtless a frequent cause of sleeplessness and other symptoms of nerve exhaustion; but when it is put very prominently forward by the patient as a cause of ill-health, a careful scrutiny should be made into his habits. It is, at least, not uncommon to find that a clerk, who is earning an assured salary, and who has no business responsibilities of his own, assigns bad trade and difficulties in business as the cause of his sufferings, when it turns out that his chief anxiety arises from the fact that he is becoming incompetent for his work, and that he has the fear of dismissal before his eyes. Patients of this class are apt to become shy and timid; they cease to mingle in society, and are often unable to hold a business interview with a well-known customer without being previously fortified by a glass of brandy. A patient, who had gradually fallen into habits of intemperance, told me that he had been greatly depressed for upwards of three years, and that he had become exceedingly timid and nervous, and could not attend to business without the frequent use of stimulants. His breath was smelling strongly of spirits, and he owned that he could not make up his mind to consult me until he had steadied his nerves by drinking a glass of brandy. "I cannot sign my name," he said, "if anyone is overlooking me." "I live in the country," he continued, "near three or four neighbours, with whom I am on friendly terms; but if I see one of them before me in the lane leading to the station I hang back so as to avoid speaking to him. I cannot endure to speak to strangers, and have a great aversion to converse with one of my most intimate friends." He had not suffered from visual hallucinations, but was very susceptible to noises, and on walking in the country he has been frequently startled by hearing what appeared to be a foot-fall behind him, when on turning round no one was present. This patient was also so absorbed in his own gloomy thoughts that in walking on the pavement he frequently stumbled against other people, and had been often in danger of being run over by cabs or other vehicles in crossing the streets. He gave way to uncontrollable outbursts of temper at home, but had no homicidal or suicidal temptations. Such temptations, however, are by no means uncommon in this stage of the symptoms. The timidity of disposition just described is frequently met with in females who give way to secret drinking. The first indication of this evil habit is often that the patient ceases to attend to her social duties; from being bright and cheerful and friendly, she becomes retiring, and refrains from visiting, and her friends on calling generally find her indisposed and not to be seen. And as the disorder increases, she becomes suspicious and distrustful of her nearest friends, and may accuse her neighbours of circulating scandal about her, or of overt acts of insult. It is in women of this type that the moral degradation produced by alcohol is best seen, for they are often mischief-makers, and their first impulse is to tell a lie, even when no purpose whatever is served by it.

The *stage of delirium* may assume the acute or chronic form. The *acute* variety, or *delirium tremens*, is mentioned as having been present in six out of the ninety cases here reported, and in many other cases it is stated that the patient had at a previous time suffered from one or more attacks. At the onset of an attack of delirium tremens the appetite fails, so that little or no food is taken for some days; the breath has a characteristic odour; the tongue is covered by a thick creamy fur, or is red and glazed, and is tremulous on protrusion; the lips tremble when the patient speaks, and the articulation is indistinct; the face is generally flushed, but occasionally pale; the pupils are usually dilated, and the conjunctivæ injected; the temperature is only slightly elevated, except in fatal cases, when it may rise to 105° F., or even to 108° or 109° F., before death; and the pulse is usually large, soft, and dicrotous. Tremors are usually present, not only in the tongue and lips, but also in the muscles of the face, head, and hands, and in some cases they are so widely diffused that the whole body trembles. The patient now becomes restless, irritable, and quarrelsome; he is agitated, and wakeful at night, and if he dozes off for a short time his sleep is disturbed by horrid dreams. Hallucinations of the special senses now make their appearance, those of sight being probably the most common. On closing his eyes for sleep the patient sees variously coloured spectra, in the midst of which grinning faces and other monstrous images appear, and as the disease advances, distinct objects are seen in broad daylight. We have seen that in the early stage of the disease the hallucinations assumed the form of pictures or photographs, which do not impose upon the patient for an instant as realities, and that in the second stage the patient is able by a simple experiment to convince himself of the unreality of his hallucinations, however vivid and corporeal they may seem at first sight; but in this stage of the disease the patient firmly believes in the reality of his hallucinations, in spite of all adverse experience and opposing testimony. The hallucinations, vivid but corrigible, in the second stage, have now become quite incorrigible. The patient sees himself surrounded by objects calculated to excite disgust or terror, such as insects, rats, cats, snakes, and monsters of variable shape, while he often fancies himself to be pursued by armed men, who threaten him with menacing gestures. He talks incessantly in an incoherent and rambling manner, and looks suspiciously under the bed, and in every corner of the room, to see that none of the imaginary beings by which he fancies himself to be surrounded are lodged there. His actions, indeed, appear to be largely determined by the nature of his hallucinations. At one time he will busy himself in endeavouring to catch the insects which crawl over his body, or he will get up and search everywhere for something that has disappeared in a corner of the room, while at another time he will dodge about in order to avert a threatened blow, or endeavour to hide in abject terror behind an article of furniture, in order to escape from some pursuing foe. The prevailing character of the patient's mind during the attack is one of terror and cowardice, but he may occasionally turn upon his attendant in the belief that the latter is plotting against him, or is about to inflict upon him some bodily injury; and although, as a rule, he is tractable to his medical attendant, he is not unfrequently violent towards his friends, and especially towards his wife. He often, indeed, labours under the delusion that his wife is in collusion with the imaginary beings by which he is surrounded, and, his jealousy being roused, he may be led to make a murderous attack upon her. An attack of acute alcoholic delirium seldom lasts more than from three or four days to a week. In most cases the patient falls into a refreshing sleep and awakes convalescent. In a few cases the temperature rises to 105° or even 108° F., and the patient dies comatose or from asthenia.

Chronic alcoholic delirium assumes many phases, and these are subject to various modifications, according to the age, sex, and hereditary tendencies of the patient, and according to the kind of alcoholic beverage in which he indulges, and the daily quantity imbibed by him. It is often stated that women have an immunity from at least the more active psychical disorders of alcoholism, but this statement is far from being true. I have attended three women within the last few months, who manifested the symptoms of chronic alcoholic delirium in their most typical form. All of them lay in bed, and presented the dropped wrists and ankles, and the other phenomena which characterise alcoholic paralysis. The first of these was a woman about fifty years of age, who had suffered in previous years from an attack of acute rheumatism, and the small joints of her hands and feet were swollen and red at the visit; but the accompanying sensory and motor phenomena bore an unmistakeable alcoholic stamp, while an undoubted history of secret drinking was obtained by her medical man. The patient spoke quite rationally at first, but she soon showed that her memory of past events was very defective. Although she had been helpless in bed for some weeks, she stated that she had been out driving on the previous day, and told me a long and somewhat incoherent tale of how she had been spirited from her home and lodged in another house, and during the whole period of her illness she uniformly asserted that she was not at her own home. She was suspicious of her attendants, and accused her nurse of poisoning her food, and of otherwise ill-treating her. She was usually moderately quiet during her medical attendant's visit, but almost immediately after he had left her room she began shouting, screaming, and struggling so far as her helpless condition would permit, and continued in this wildly maniacal condition for three or four weeks, with only an occasional intermission from exhaustion, or when sleep was procured by a strong dose of bromide of potassium and chloral hydrate, or of chloramid. After some weeks she became calmer, and gradually passed into a condition of dementia, from which she has made only an imperfect recovery. This patient laboured for weeks under the delusion that she had undergone a severe operation, and that a baby had been extracted from her by cutting through the abdominal wall. She also harboured for a long time the delusion that the baby was in bed with her. Throughout the whole course of the psychical disorder her memory, especially for recent events, was found to be very defective, and her other intellectual powers had, doubtless, sustained a proportionate damage. Failure of memory appears, indeed, to be by far the most common mental symptom observed in the chronic delirium of alcohol, being present 25 times in the 90 cases of paralysis here collected. Accompanying this defect of memory there is usually associated a peculiar disorder in the appreciation of time and locality. The patient mentioned above never could tell the correct time of day, or the day of the week, and she always maintained that she was not at her own home, and in respect of these delusions she may be regarded as only a type of most other cases. She was the subject of very vivid visual hallucinations, and these consisted for the most part of various forms of fish. Sometimes she demanded of her attendants—"Why do you not take away that fish and cook it?" At other times she saw crocodiles and other huge monsters approach her with gaping mouths, and in menacing attitudes. She does not appear to have had any aural hallucinations, although in other cases these predominate, and the patient may then hear strains of music or songs, or still more frequently oaths and blasphemous curses. Illusions of the senses are also not uncommon. The patient, for instance, imagines that the footstool under the table is a dog, or that the pincushion is a rat, and so on. The second of the three women suffering from chronic alcoholic delirium observed by me this year had passed into a state of more or less calm and dementia when I visited her, but on enquiring into the character of her mental disorder I found that she had suffered for many weeks from wild delirium, hallucinations, and delusions similar in every essential particular to those observed in the first case. Her daughter told me that throughout her illness her memory was very defective, and she always fancied herself away from home, and was very suspicious of every one surrounding her. Her daughter, on being asked if her mother had complained of seeing animals or fish about her bed, said, "Oh, yes, every little fold in the bedclothes she imagined to be a fish." In the third case—the landlady of a public-house, about thirty-five years of age—the mental symptoms had pursued a somewhat acute course, and she had already become fairly calm and rational when I saw her. She had been removed from her own home to another house, in order the better to protect her from having access to more drink, but she could not be persuaded that she was not still lying at her home. Her memory for recent events was very defective, but on being questioned as to hallucinations she laughingly denied having seen any animals or fish. Her medical attendant, however, told me that in the early part of her mental disorder she was constantly talking of seeing cats and fish. The lighting pains and other sensory disorders of alcoholic paralysis appear to give rise to special delusions. Some patients imagine that their skin is covered by vermin and other insects, while it is a very common delusion of alcoholic subjects to believe that some one is torturing them by an electric battery.

In other cases the patient is morose and the subject of a profound melancholia, with delusions of persecution, but the mental depression is apt to be interrupted by periods of great excitement. Such patients are often very treacherous, and may suddenly give way to a suicidal, or more often to a homicidal, impulse. At other times the patient is seized with a succession of epileptic fits, and may pass more or less suddenly into a condition of dementia, with or without hallucinations and delusions. Some months ago I saw a man who had two or three epileptic attacks whilst drinking freely, and immediately afterwards it was found that his memory was very defective. During the day he was fairly rational, but at night he thought his bedroom was invaded by a large number of men, whom he saw distinctly standing by his bed side. He acknowledged that these men seemed to pass through the locked door, and to disappear mysteriously behind the curtains and furniture; but even in his most rational moments, he could not be persuaded that they were not realities. He thought that his wife was in collusion with them, and accused her of infidelity.

In women the stage of delirium is very unobtrusive, and many of them pass silently and quietly into a state of almost hopeless dementia, without its being preceded by obvious delirium, epileptic attack, or any form of excitement. It is hardly necessary for me to state that no attempt is made here to describe the various forms of chronic insanity met with in alcoholic subjects; the reader must seek for information on this part of the subject in works on psychological medicine.

The stage of alcoholic *dementia*, once established, is very characteristic. The symptoms were well marked in J. C. (Case 5), and as they have already been described they need not be reproduced here. The memory is almost a complete blank, and a patient, who may be lying helpless in bed for weeks, will give a circumstantial account of how he had been out in the morning to fetch beer, or how he had walked out and visited several public-houses with a boon companion. An Infirmary patient of my own, who lay helpless in bed for nine weeks, narrated, day by day, to us, how he had been out walking on the same morning. To the usual question of "Where have you been to-day?" he would reply, "Oh! I have been out to the pier. It was blowing quite fresh, but it has done me good." "Have you had anything to drink?" "Oh! yes; I met a friend—I forget his name, but I know him quite well—and we went to a public-house and had three-pennyworth of whisky each." On another day he would describe himself as having been walking in Whalley, of which district, I believe, he was a native, and he never failed to praise the beauty of the country, or to meet a friend with whom he had the inevitable three-pennyworth of whisky. This man was also accustomed, for many weeks, to tell us he had a baby in bed with him, and, at first, he was not a bit abashed when it was pointed out to him that there was no baby in bed, but as his mental condition somewhat improved he met us with the statement, "Oh! its mother has taken it." This man recovered from the paralysis and was sent to the Convalescent Hospital, at Cheadle, but although he ceased to tell us of his morning adventures, his memory remained very defective, and he was quite unfitted to return to his previous avocation of clerk in a commercial house.

(b) The Paralysis of Carbon Monoxide Poisoning.

In the following two cases the paralysis appears to have been caused by the inhalation of carbon monoxide gas:—

Case 1.—J. E., aged 50 years, was admitted to the Manchester Royal Infirmary on June 20th, 1889.

The patient is a well-built man, and his face is somewhat bronzed by exposure to air and sun, but on closer inspection it is seen that his lips and conjunctivæ are very pale and anæmic. He states that up to the beginning of this year he never was absent a day from his work from illness, and was very robust and healthy. He has always been in easy circumstances, well-clothed and well-fed, never suffered from any venereal affection, and never been addicted to excess in drinking. About the beginning of 1887 he began to work a new patent for making gas for lighting purposes. He had to stand over the retorts while replenishing them with coal, and consequently had to inhale a great deal of the gas which issued from them, and was much exposed to the fumes whilst clearing the furnaces. In July, 1887, he began to suffer from a severe dull pain in the right hypochondriac region, and at the same time complained of so much shortness of breath on exertion, that he had to stop several times, whilst walking home at night, to recover his breath. These symptoms increased to such a degree that he was soon compelled to desist from his work, but after being under the treatment of a medical man for six weeks, he was able to return to his former occupation. He soon, however, began to suffer from his former symptoms, and after some months he experienced shooting pains in the legs and about the shoulders and arms. His hands and feet were quite numb, especially in the morning, and he also suffered greatly from cramps of the calves of the legs at night, and had often, in consequence, to jump out of bed and plant his feet firmly on the floor. His legs also began to swell about the ankles. The swelling was, at first, soft and pitted on pressure, but after a time it extended up to the knees and did not yield to the pressure of the finger. In the beginning of February, 1889, the patient found that his right hand was quite numb and dead each morning; the fingers were bent into the palm, and he was unable to open his right hand until he stretched the fingers by the other hand. After rubbing the right hand for some time, "to get up the circulation," the numbness partially disappeared, and he was able to move the fingers, but on the following morning his hand was as numb and powerless as before. The left hand was soon similarly affected, and his legs became so feeble that he was hardly able to stand and once more he was compelled to desist from his work. The feeling of weakness of the lower extremities and the powerlessness of his hands gradually increased in intensity after he left off work, and at the end of some weeks he could hardly stand, and was quite unable to button his clothes. After a time improvement set in very slowly, but as he was not recovering his strength as fast as he expected he entered the Infirmary as an in-patient.

Present Condition.—On walking the patient manifests the most characteristic form of the "high stepping gait," caused by a drop of the toes of the advancing foot, owing to paralysis of the anterior muscles of the leg. When he sits in a chair with the feet planted on the ground he is quite unable to raise the toes of either foot, and he is also unable to stand on the tips of his toes. On lying on his back in bed he has well marked double ankle drop, and the toes are hyper-extended at the metatarso-phalangeal and flexed at the phalangeal joints, but he can move the feet at the ankles, and the toes at all the joints by voluntary effort, although but feebly. His hands are not dropped at the wrists, but the power of extension is feeble, and he cannot oppose the tips of the little finger and thumb of either hand without flexing the thumb at the phalangeal joint. The thenar and hypothenar eminences are slightly flattened, and the backs of the hands are somewhat grooved, but the muscular masses of the forearms and of the lower extremities are not atrophied to any perceptible degree. The face is destitute of wrinkles and expressionless, and the left orbicularis palpebrarum is weaker than the right one. The tensor tarsi of the left side is also feeble, so that the lower eyelid is somewhat everted, and the tears flow over the cheek. The pupils are somewhat dilated, but they are equal and react to light and accommodation. The ocular movements are normal. The toes are almost completely insensitive to the pricking of a pin, and the backs of the feet also manifest a considerable degree of anæsthesia, but a prick is readily felt over the surfaces of both legs, and there is no retardation of sensory conduction. Two points have to be separated about nine inches before they are felt as two over the external surfaces of the legs. The sense of temperature does not appear to be much diminished, but muscular sensibility, as tested by the power of the patient to judge of the position of his lower extremities with closed eyes, is somewhat diminished. The plantar nerves are very sensitive to pressure, especially at their points of bifurcation to form the digital branches, and the popliteal nerves are also very painful on pressure. The special senses are unaffected, and the optic discs are found to be normal on ophthalmoscopic examination. The patellar-tendon reactions are absent in both legs, but the cutaneous reflexes are preserved. The patient at one time saw faces and scenes like pictures on closing his eyes for sleep, but these have now ceased to trouble him. He is still, however, very sleepless, and when he does fall asleep his rest is much disturbed by horrid dreams. The cardiac dulness is somewhat enlarged, and the second sound is accentuated at the base. The pulse beats 90 in the minute and is regular, but very compressible. The legs are somewhat swollen, and pit on pressure of the finger. The urine is free from albumen and sugar. The number of corpuscles to each mm. of blood is 3,920,000. The hæmoglobin measured by the hæmoglobinometer is equal to 70 per cent. The lungs are normal, but the patient is very breathless on the slightest exertion. The patient's appetite is excellent, and his digestion is normally performed. All the muscles react to a weak faradic current, with the exception of the peronei muscles of both sides which only give a minimum contraction, and the proper extensors of the big toes which fail to react to the strongest current. The following galvanic reactions are obtained :—

			RIGHT.			LEFT.		
			K.S.C.		A.S.C.	K.S.C.		A.S.C.
Orbicularis palpebrarum	Cells	Leclanché	15		20	15		20
Levator labii sup.	„	„	15		20	15		20
Levator anguli oris	„	„	15		20	20	>	20
Tibialis anticus	„	„	20	<	20	25		15
Extensor longus digitorum	„	„	25	<	25	30		20
Peronei	„	„	25	<	25	30		25
Extensor proprius pollicis	„	„	25		25	30	<	30
Rectus femoris	„	„	—		—	40		30

Aug. 1.—J. E. has been treated since his admission by the administration of iron, good diet, rest in bed, and massage, but although his general health has improved considerably, motor power has only returned very slowly to the muscles most affected, and he still manifests a well-marked "high-stepping gait," while he cannot fully approximate the tips of the little finger and thumb in either hand. He was now sent to the Convalescent Hospital at Cheadle, where he remained another month. During his residence there he regained further motor power, and the patellar tendon reactions reappeared, but when last seen, about the end of December—six months after his admission as an in-patient, and nearly twelve months after he gave up work—he was unable to resume any occupation, although the paralysed muscles had regained so much of their motor power as to render it certain that eventually he would make a complete recovery.

Case 2.—W. H. G., aged 43 years, entered the Manchester Royal Infirmary October 25th, 1889. The patient is a furnaceman by occupation, and is much exposed to the fumes of burning coal whilst filling the furnace, and especially whilst cleaning the furnace by raking out the hot cinders. He has always enjoyed excellent health, and has been a very strong man up to the date of his present illness, which began about eighteen months ago. The first symptom which attracted his attention was a feeling of general weakness; his knees felt to give way under him, and his legs felt as heavy as lead. He had, indeed, suffered for the previous twelve months from numbness and tingling of the hands and feet, but he took no notice of these symptoms until the weakness began. He also felt giddy whilst walking, and experienced much difficulty in breathing whilst walking up a hill and in going up stairs. His appetite failed, and he felt listless and depressed, and had no inclination to go out in the evening after he returned from work, and at night his rest was broken by horrid dreams. In the morning he awoke unrefreshed and indisposed to return to his work. About twelve months ago he had an attack of diarrhœa, which lasted several days, and was obliged to remain at home in consequence of it, and when in a few days the diarrhœa ceased, he felt so weak that he was quite unable to return to work. His face was of a yellow colour, and he was supposed to be suffering from liver disease, but instead of getting better he became day by day weaker, until in a few weeks he could not button his clothes, and was scarcely able to stand. He is a married man, and is the father of seven healthy children. His wife had no miscarriages or dead-born children. He has never contracted syphilis, and has always been a temperate man.

Present Condition.—The patient is a powerfully-built man, but his face has a sallow colour, and his lips and conjunctivæ are pale and anæmic. On standing, with feet approximated and eyes closed, the patient sways from side to side; he cannot stand on the tips of his toes, and in walking the toes of the advancing foot drop, so that the gait is "high stepping." The patient when sitting is unable to raise by voluntary effort the ball of either foot from the floor. The feet, when unsupported, drop at the ankles; and the toes are hyper-extended at the metatarso-phalangeal and flexed at the phalangeal joints. The patient's grasp is feeble, and he experiences great difficulty in performing delicate movements with his hands, such as picking up a pin and buttoning his clothes. The face is destitute of wrinkles and expressionless, but there is no evidence of decided paralysis of any of the facial muscles. The patient suffered from diplopia some time ago, but this symptom has now disappeared. He has often experienced some difficulty in commencing to micturate; on one occasion his bladder has had to be emptied by means of the catheter, and he has occasionally suffered from dribbling of urine.

The muscles of the proximal segments of the extremities and of the body generally are found to be remarkably deficient in strength when tested by opposing actions.

The patient complains of tingling of the hands and numbness of the finger tips, and of burning sensations in the soles of the feet, and a dead, numb feeling in the toes, but there are no marked sensory disorders to objective examination. One of the spinous processes at mid-dorsal region is tender to pressure, and the patient complains of a girdle pain passing round the body at this level. The special senses are unaffected. The pupils react to light and accommodation, and the optic discs are found to be normal on ophthalmoscopic examination. On raising his head suddenly from the horizontal position he is much distressed by dizziness, and when he is erect an attack is induced if he raises his arms above the horizontal level. The cutaneous reflexes are active, but the patellar tendon reactions are lost, and there is no ankle clonus.

The cardiac dulness is somewhat enlarged; the apex beat is indistinct; the first sound at the apex is impure, and the second at the base accentuated; and the pulse is 85, regular, and very compressible. There is no œdema of the ankles. The patient complains of pain at the pit of the stomach, heartburn, and flatulence; his tongue is coated, the bowels are constipated, and he has no relish for his food.

The urine is high coloured, free from sediment, albumen and sugar, and the specific gravity is 1015. The temperature has remained throughout normal.

The following reactions were obtained to electrical examination. All the affected nerves and muscles reacted to a weak faradic current.

The galvanic current gave the following reactions :—

				Right.		Left.	
				K.S.C.	A.S.C.	K.S.C.	A.S.C.
Arm.—	Extensor communis digit...	Cells	Leclanché......	20	25	20	25
	Extensor carpi ulnaris ...	„	„	20	25	10	15
	Flexor digitorum sublimis	„	„	15	20	15	20
	Thenar muscles	„	„	20	25	15	20
	Hypothenar muscles	„	„	20	25	20	25
	First interosseous	„	„	15	20	15	20
Leg.—	Tibialis anticus	„	„	20	25	20	> 20
	Extensor longus digitorum	„	„	20	25	15	20
	Peronei	„	„	20	25	25	> 25
	Rectus femoris...............	„	„	25	30	25	30
	Gastrocnemius...............	„	„	25	30	25	30

The patient was treated by the administration of iron, good feeding, rest in bed, and massage.

Nov. 13.—The patient has improved both in motor power and general health since his admission, and he was now sent to the Convalescent Hospital at Cheadle.

In the two cases just reported it is open to question whether or not they were real examples of poisoning by carbon monoxide. In the first place the patients were exposed to fumes, which in addition to carbon monoxide would, doubtless, contain a predominating proportion of carbonic acid, various forms of carburetted hydrogen and other compound gases, so that even if it could be proved that these fumes were the cause of the paralysis, it would still remain doubtful as to the part which the carbon monoxide had played in the production of the symptoms. Into the chemical part of the question, however, we do not enter. We shall take it as already proved that of all these gases the carbon monoxide is incomparably the most deleterious, and also that it acts, as Bernard pointed out, by forming a stable compound with the hæmoglobin of the blood. The question now is, was exposure to the fumes of burning charcoal or coal the cause of the neuritis in these two cases? The following is a summary of the evidence in favour of an affirmative answer to this question. The patients were undoubtedly much exposed to the action of these fumes during the whole of the working day, and were unduly exposed to their action when cleaning out the retorts. The first symptoms of which they complained were pain in the hypochondriac region near the line of the attachment of the diaphragm, shortness of breath, and dyspnœa on exertion, and these are the symptoms that a poison like carbon monoxide, which deprives the hæmoglobin of its function of aerating the blood, might be expected to produce, and, as a matter of fact, are the symptoms which are known from clinical records to be caused by this poison.

Again, these patients, although presenting all the usual symptoms of peripheral neuritis, yet were both temperate men; they had not suffered from sore throat to indicate diphtheria, had not contracted syphilis, were not exposed to lead, arsenic, or other metallic poison, and were not the subjects of gout, rheumatism, or diabetes; in short, the closest scrutiny into their previous history failed to discover an exposure to the action of any kind of poison capable of producing neuritis, with the exception of the fumes of burning coal. That the carbon monoxide present in these fumes had produced a deleterious effect upon these men was rendered highly probable by the fact that, although both had evidently been powerful, able-bodied, and otherwise healthy men in the prime of life, yet they manifested a degree of anæmia which could hardly be explained except on the supposition that repeated exposure to this noxious agent had caused great destruction of the hæmoglobin.

The following case, reported by Leudet,* is such a good example ot the immediate and remote symptoms caused by exposure to charcoal fumes that we make no apology for quoting it in detail.

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* Leudet (E.). "Sur les troubles des nerfs périphériques et surtout des nerfs vaso-moteurs consécutifs à l'asphyxie par la vapeur de charbon."—*Archives Générales de Médecine*, VI. Série, Tome V., Paris, 1865, p. 516.

Alexander Holding, aged 30 years, a stoker on board a steam-tug, entered the Hôtel Dieu on November 20th, 1863, and was placed under the care of Dr. Leudet. On the night of the 19th Holding, being in good health, retired to his cabin, closed the hatches, and, in order to warm himself, lighted, by means of a little oil, some charcoal in a pan. At five o'clock on the following morning one of his companions found him in an insensible condition, while the bedclothes were not disordered, and the pan was not upset. On the morning of the 20th Holding was quite unconscious, comatose, and insensible to pricking. His face was pale, and the posterior and external aspect of each forearm over the distribution of the musculo-spiral nerve was of a red colour, but without any tumefaction of the subjacent cellular tissue. The clothing which covered the forearms was intact and without any trace of burning. A spot of redness, similar to that observed on the forearms, and also without being attended by swelling, was noticed on the right temple near to the external margin of the orbit. The extremities were cold, and the pupils were dilated, while the pulse was small and frequent. Mustard plasters placed over the calves of the legs did not give rise to any pain, and electrisation of the extremities did not evoke muscular contraction. A few hours later Holding ceased to be comatose; he opened his eyes occasionally, but he soon closed them, and he did not reply to any questions. In the evening he became again comatose. Blisters were now applied to the calves of the legs, and 125 grms. of blood were taken from the arm. The blood withdrawn was of a rosy tint, and did not coagulate.

On the 21st the coma persisted, but without stertor; the face was pale, the pulse beat 128 in a minute, and there was complete resolution of the extremities. A few herpetic vesicles now appeared over the right temple, but the redness became fainter. The redness of the right forearm, however, became increased in intensity, but that on the left forearm had almost entirely disappeared. The urine was passed involuntarily. An injection of a solution of salt, to which 15 minims of tincture of colocynth was added, was now administered. The coma persisted until the night of the 21st, but on the morning of the 22nd he was found quite sensible, the skin was bathed with sweat, and the pulse beat 72 in the minute. He had no recollection of any event which occurred since his admission to the hospital. He did not suffer any pain, but he complained of a feeling of numbness of the three last digits of the right hand, and he was unable to extend them fully, while they were maintained in a state of exaggerated flexion. In the two first digits of the right hand the movements of flexion and extension were almost normal. Small groups of herpetic vesicles, each about the size of a pin's head, appeared on the anterior and internal aspect of the forearm, the redness already mentioned lying to the outside of them. Each group of vesicles is oblong in form, being 3 cm. in length and 1½ cm. in breadth. The underlying skin is of a uniform red colour and the subjacent cellular tissue is somewhat infiltrated. The sensibility of this part, as indeed, of the whole arm, is unaffected. An eschar, about the size of the palm of the hand, appeared over the inferior part of the sacrum; it was of a brownish colour, and there was no redness of the skin surrounding it. Holding continued in the same condition up to Nov. 30th; he was now quite sensible, and there was no fever, but extension of the three last digits of the right hand was still impossible. The movements of flexion of the digits and all the movements of the hand and forearm were maintained, and sensibility was intact. The herpetic vesicles dried up rapidly, and the redness of the right forearm became fainter. The eschar on the sacrum became surrounded by a red aureole, and the gangrenous portion now began to separate. The appetite was normal, and there was no dyspnœa or paralysis of the lower extremities.

On December 1st about twenty groups of herpetic vesicles, each about the size of a pin point, appeared on the posterior aspect of the right thigh. The groups were mostly distributed along the course of the sciatic nerve, but a few of them ascended like a vertical band over the right buttock. The subjacent skin was of a slightly red colour. The patient complained of a little local pain, but there was no formication or other sensory or motor disorder of the thigh or leg.

From December 5th to 20th the patient improved greatly in his general health. He now only complained of some slight pain about the eschar on the sacrum, the gangrenous portion of which was nearly detached, leaving a healthy granulating surface. The condition of the right forearm had not undergone much alteration; the local swelling and redness had diminished somewhat, but the patient was still unable to move the three last digits of the right hand, and even the movements of the index finger and thumb were restricted and feebler than normal. Electrical stimulation of the nerves and extensor muscles of the three paralysed digits failed to evoke either contraction or pain. The electrical reactions on the anterior aspect of the forearm and of all the nerves and muscles of the upper arm were normal. On December 24th the patient was discharged.

It will be seen that in this case the paralysis is almost exclusively limited to the extensors of the three ulnar fingers of the right hand. In a case reported by Rendu* the paralysis assumed the hemiplegic form.

A woman, aged 31 years, being exposed, while ironing linen, to the fumes from a stove, lost consciousness and was found comatose. On awakening on the following morning, the right half of the body was paralysed, but without aphasia. A month later she was admitted to the hospital, and it was then found that the muscles of the right half of the face, including the orbicularis palpebrarum, the extensors, and to a less degree, the flexors of the forearm, and the extensors of the leg were paralysed. The inferior two-thirds of the forearm and of the leg were anæsthetic, and so also was the skin of the little finger and that over the inner surface of the index finger of the left hand. The patellar tendon reactions were exaggerated. The electrical contractility of the affected muscles was lost. A soft, circumscribed swelling appeared over the anterior surface of the right forearm, and the anæsthetic portions of the skin presented a glossy appearance, and were covered by sweat. Notwithstanding the hemiplegic distribution of the paralysis, the author believed, and doubtless with justice, that the symptoms were caused, not by a central lesion, but by a peripheral neutritis. A case is reported by Knapp,[*] in which a patient after exposure to the fumes of burning charcoal was found in an unconscious condition, and for ten days his life was despaired of. On recovering consciousness, it was found that all the external and internal muscles of both eyes were completely paralysed, so that not only all movements of the eyeballs were impossible, but a considerable degree of paralytic exophthalmos was present, and the power of accommodation for near vision was lost. In the course of two months considerable improvement took place, the ciliary muscles and the sphincters of the pupil recovered, and the superior, external, and inferior recti were also restored to some extent, but the internal recti remained paralysed, and the development of a persistent double external squint had to be overcome by tenotomy of the two external recti muscles. In a man whose case is recorded by Litten,[†] the right arm, twenty hours after he had recovered consciousness, became the subject of a brawny swelling, which extended from the fingers to above the shoulder. The whole of the arm was paralysed and completely anæsthetic, while the reflex and electrical irritability of both nerves were lost. The back of the hand was covered by pemphigus bullæ. The author believes that the symptoms were caused by disease of the brachial plexus caused by hæmorrhagic infiltration.

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* Rendu (H.) "Intoxication par la vapeur de charbon. Paralysie consécutive interressant la face du côté droit, ainsi que les extenseurs de l'avant bras et du pied du même côté. Guérison lente."—*Union Méd.*, Paris, 1882, Nos. 33 and 34, . 386 et 397, et *Gazette hebdomadaire*, June 20, 1882.

In other cases the paralysis is widely distributed. Leudet[‡] reported a case of acute ascending paralysis as occurring after exposure to the fumes of burning charcoal, and which has already been mentioned in these pages. The patient was a man of 51 years of age, who attempted to commit suicide, while in a drunken condition, by inhaling the vapour of burning charcoal. He was conveyed in an insensible condition to the Hôtel Dieu de Rouen, and on recovering consciousness two days later he complained of a heavy pain which radiated to the right buttock and was aggravated by pressure over the sciatic nerve. He complained from the first of great general feebleness, but it was not until seventeen days after the exposure to the poison that the right leg became completely paralysed, and the left was similarly affected on the following day. The paralysis now gradually extended, and successively invaded the muscles of the upper extremities and face and those of deglutition and respiration, and the patient died in a state of delirium 30 days after exposure to the charcoal fumes. At the autopsy the bases of both lungs were engorged. The nervous centres were normal. The right sciatic nerve at its point of emergence from the pelvis was one-third larger than the left at the corresponding part. The sheath of the swollen nerve was injected and thickened, and its fibres were separated by a growth of fibro-cellular tissue, which surrounded and compressed the nerve fibres. The swollen part of the nerve was only about the breadth of the thumb in vertical extent, and below this part the nerve appeared to have been healthy. The value of this case, however, is diminished by the fact that the patient was a great drunkard, and it is, therefore, impossible to determine how much of the symptoms is due to the alcoholic excesses, and how much to the carbon-monoxide poisoning. A curious case of poisoning by exposure to illuminating gas in a closed room is recorded by Backer,[*] in which the after effects simulated the symptoms of disseminated sclerosis. The patient was found in a state of unconsciousness and cyanosis, but after artificial respiration had been used for two hours the breathing was re-established. At the same time fibrillary contractions appeared in the muscles of the left half of the body, and these increased in eight hours to strong muscular spasms, which were evoked on the slightest contact. When seen three months later the patient suffered from tremors on voluntary movements, fibrillary contractions of the tongue, slow and stammering speech, tremulous and unsteady handwriting, and a deviation to the right of the uvula. The right patellar tendon reaction was more lively than the left. He also suffered from scintillating vision and vertigo, while on ophthalmoscopic examination the retinal veins were found to be congested, and a small exudation was observed on the inferior margin of the right pupil. His memory was impaired, and he had no recollection of the events which occurred for a considerable time after his exposure to the poison. He recovered completely five months from the commencement. This case departs so much from the ordinary type that it is doubtful whether or not it is to be regarded as an example of multiple neuritis, but there is at least nothing in the nature of the symptoms to negative this supposition. The fact that complete recovery took place in five months suffices to show that the case was not a true example of disseminated sclerosis.

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[*] Knapp (H.). "Ein Fall von Parese der Augenmuskeln durch Kohlendunstvergiftung."—*Archiv für Augenheilkunde*, Bd. IX., 1888, p. 229.

[†] Litten. "Ein seltener Fall von Kohlenoxydvergiftung."—*Deutsche med. Wochenschrift*, 1889, No. 5.

[‡] Leudet (E.). *Loc. cit.*, p. 525.

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[*] Bäcker (E.) "Ueber Nachkrankheiten der Kohlenoxydgasvergiftung, speciell über einen unter dem Bilde der multiplen disseminirten Sklerose des Centralnervensystems verlaufenden Fall."—*Deutsche med. Wochenschrift*, 1889, Nos. 26 u. 28.

In addition to sensory and motor disturbances, well-marked vasomotor and trophic disorders result from exposure to charcoal fumes or carbon monoxide gas. In the case of Holding, patches of redness appeared on the forearms, which could not have been caused by burning, as the clothes over the affected part were intact. Crops of herpetic vesicles appeared over the right forearm, and the subjacent subcutaneous tissue was infiltrated. Similar crops of vesicles appeared on the right buttock and along the posterior aspect of the thigh, and a large eschar formed on the sacrum. Another case is reported by Leudet,[*] in which the patient had during the night exposed himself to the fumes of burning charcoal with the view of committing suicide. He recovered consciousness the following morning, although still somewhat somnolent; but on the eleventh day after exposure to the fumes a crop of herpes appeared on the left half of the face, the vesicles being situated over the forehead, the cheek and the chin, along the distribution of the supra-orbital, infra-orbital, and mental branches of the trigeminal nerve respectively. The patient appeared to improve during the following fortnight, but at the end of this time symptoms of pulmonary congestion supervened and a blister was applied over the posterior and lateral aspect of the chest. The blistered surface became red and inflamed, and a dark gangrenous line appeared about the level of the inferior angle of the scapula, showing the want of vitality of the tissues. The patient died a few days later, but there was no autopsy. Other remarkable examples of trophic disorders after poisoning by carbon fumes are reported by Hasse.[†]

Five soldiers were exposed to the fumes disengaged during the combustion of oil in a stove, the valves of which were closed. Two were found dead, a third died in convulsions a few hours later, and the two remaining survived for some time longer. The first of these remained in an unconscious condition for eight days, and was completely paralysed until the twelfth day, when he died. On the sixth day the whole surface of the body became covered with pemphigus bullæ, which, on bursting, left the subjacent surface in a raw and ulcerated condition. Two days later a fresh crop of pemphigus bullæ appeared, and likewise an eschar on the sacrum, which was the seat of a profuse suppuration. The extremities were completely paralysed, and so also was the bladder, and the urine was ammoniacal and contained sugar. The second patient, who survived, regained consciousness at the end of 24 hours after exposure to the poison, but he remained very feeble and depressed, there was incomplete paralysis of the bladder, and after being only three days in bed a very large abscess appeared over the chest and the right buttock. In a case reported by Alberti[*] there was, in addition to paralysis of the right leg, extensive gangrenous destruction of the muscles of the neck.

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[*] Leudet (E.). *Loc. cit.*, p. 521.

[†] Hasse. *Preus. Ver. Zeit.*, N. S., Bd. II., 1859, p. 35. Abstr. in *Canstatt's Jahresb*, Bd. V., 1860, s. 102. Quoted by Leudet, *Loc. cit.*, p. 520. See also Arnozan et Dalidet, "Empoisonnement par les vapeurs de charbon. Eschares, Névrites."—*Journal de Médecine de Bordeaux.* Bordeaux, 1883, p. 30.

In one of the cases reported by Hasse, already mentioned, the urine was ammoniacal, and contained sugar, and saccharine urine has also been observed by Ollivier,[†] Kahler,[‡] and Jacksch.[§]

The special senses are not often affected, but Comby[¶] reported a case of carbon monoxide poisoning, in which a right-sided hemiplegia was accompanied by blindness, but as the patient recovered it is probable that the lesion of the visual nervous apparatus was a functional one. In the cases just reported by myself, great anæmia was a very prominent symptom, but it would be out of place to discuss here the chemical affinity which carbon monoxide has for the hæmoglobin, and the serious consequences which result from it.

The *psychical* disorders which result from poisoning by carbon monoxide are very numerous and important. Many of these disorders, however, occur during the early stage of poisoning, and it does not fall within the scope of these papers to describe them. The early symptoms were excellently delineated many years ago by Golding Bird,[**] and a description of them will be found in any standard work on toxicology. A curious example of subconscious actions during the early stage of carbon monoxide poisoning was reported by Leudet.[††] The patient was awakened during the night by the flames and smell of burning from a fire which broke out in the adjoining apartment. He arose and pointed out the way of escape to his sleeping companion; but instead of escaping himself he crept under his bed, where he was found soon afterwards in an unconscious condition. In the cases which came under my own observation, the persistent mental disorders consisted of depression of spirits, apathy, and inability to concentrate the thoughts upon any subject; but cases have been described by Barthélemy and Magnan[*] in which the patient became permanently feeble-minded, and one case in which general paralysis developed soon after exposure to the poison. A case in which amnesia supervened, after exposure to carbon monoxide gas, is mentioned by Briaud,[†] and he states that the defect of memory was very similar to that met with in cases of chronic alcoholism.

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[*] Alberti. "Ausgedehnte Gangrän der Halsmusculatur und Lähmung des rechten Beins in Folge von Neuritis ischiadica nach Kohlenoxydgasvergiftung."—*Deutsche Zeitschrift für Chirurgie*, 1884, p. 476.

[†] Ollivier. "De la glycosurie dans l'asphyxie par les vapeurs du charbon."—*Archives Générales de Médecine*, Tome CXLIV., Paris, 1879, p. 513.

[‡] Kahler (O.). "Erfahrungen über die Glykosurie bei Kohlenoxydvergiftungen."—*Prager med. Wochenschrift*, Nos. 48 u 49.

[§] Jacksch (R. v.). "Ueber Glykosurie bei Kohlenoxydvergiftung."—*Prager med. Wochenschrift*, 1882, No. 17.

[¶] Comby (J). "Asphyxie par les vapeurs du charbon."—*Archives Générales de Médecine*, Tome CXLIV., Paris, 1879, p. 513.

[**] Bird (Golding). "Observations on poisoning by the vapours of burning charcoal and Coals."—*Guy's Hospital Reports*, Vol. IV., London, 1839, p. 92.

[††] Leudet (E.). "Note sur quelques accidents par l'asphysie par la vapeur de charbon, les phénoménes d'inconscience, de paralysie péripheriques."—*Bull. de l'Academie de Médecine*, 1883, p. 1073.

With regard to the pathological anatomy of the disease, we have already seen that in the fatal case of acute ascending paralysis following exposure to charcoal fumes, reported by Leudet, he found decided evidences of neuritis of the right sciatic nerve. The patient, however, had been a chronic drunkard, and had been indulging to excess at the time of his exposure to the poison, so that it is doubtful if the subsequent paralysis is to be attributed to the inhalation of the poisonous gas. No further anatomical confirmation of the theory of neuritis has as yet been forthcoming, but the clinical phenomena clearly indicate that the cause of the paralysis is to be found in a lesion of the peripheral nerves, and not of the nerve centres. Poelchen[‡] reported a case of softening of the brain after carbon monoxide poisoning, and Gnauck[§] a case of dementia, in which symmetrically placed spots of softening were found in the lenticular nuclei, but these lesions would not account for the kind of paralysis met with in this form of poisoning. The experimental researches of Heineke[‖] seem to show that in poisoning by carbon fumes or by illuminating gas, thrombosis of a large number of the arterioles of the body occurs, and this process might account for the softening of the brain found in the above cases. It is also possible that the poison acts primarily on the arterioles of the peripheral nerves, and not on the nerve fibres themselves, but we cannot discuss the theory of the process further at present.

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[*] Barthélemy et Magnan. "Intoxication par les vapeurs de charbon, coma, convulsions toniques et cloniques subintrantes, contractures consecutives, eruption acneiforme du tronc et de la face; troubles intellectuelles graves; dêlire, colapsus, hébétudes et amnésie persistante de ces derniers phénomenes."—*Ann. d'hygiene pub.*, 1881, p. 406.

[†] Briaud. "Congrès international mentale à Paris."—*Progrès Médical*, Nos. 32 and 33, Paris, 1889.

[‡] Poelchen. "Gehirnerweichung nach Vergiftung mit Kohlendunst," *Berl. klin. Wochenschrift*, Berlin, 1882, p. 396.

[§] Gnauck, R. "Ein Fall von Verrücktheit nach Kohlenoxydvergiftung." *Charité-annalen*, Berlin, 1883, p. 402; und "Stupor nach Kohlenoxydvergiftung," *Ibid*, p. 409.

[‖] Heineke (W.). "Die Fermentintoxication und deren Beziehung zur Sublimat-und Leuchtgasvergiftung."—*Deutsches Archiv für klin. Medicin*, Bd. XLII., Leipzig, 1888, p. 147.

With regard to prognosis, it is generally said that, if the patient survive, recovery from the paralysis soon takes place. In my own cases, however, the paralytic symptoms proved singularly rebellious to treatment, notwithstanding the fact that the affected nerves and muscles reacted in a normal, or nearly normal, manner to faradic and galvanic stimulation. In one case reported by Leudet, and another by Rendu, the electrical contractility of the paralysed muscles was lost, so that a profound degree of paralysis must have been present. In the case of the implication of the oculo-motor muscles, reported by Knapp, the paralysis proved very rebellious to treatment. It would appear therefore that the prospect of recovery from the consecutive paralysis is by no means so favourable as has been supposed, and it is possible that the duration of the paralysis is increased rather than diminished when the paralysis results, as in my own cases, from a chronic and repeated, and not from a single exposure to the action of the poison.

Carbonic acid may possibly of itself suffice to give rise to multiple neuritis. A case of œdema of the glottis is mentioned by Leudet,* in which the patient, after being on the verge of death for fifteen hours, was rescued by tracheotomy, and forty-eight hours later a large eschar appeared on the sacrum. The author remarks, "it would thus appear that the prolonged arrest of the oxygenation of the blood exercises a deleterious influence upon the nutrition of peripheral parts." In cases dying from slow asphyxia, I have frequently found loss of the tendon reactions, and other indications of a moderate degree of peripheral neuritis; but in many of these cases the patients died from cardiac obstructive diseases or chronic bronchitis, and it was found impossible to exclude the action of alcohol. In one case of œdema of the glottis, which came under my observation, in a young man of temperate habits, the patellar tendon reactions were absent; but it was difficult to prove any special paralysis amidst the general feebleness which presaged the fatal issue.

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* Leudet (E.) *Moniteur des Hôpitaux*, 1856, et *Loc. cit.*, p. 518.

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(*c*) THE PARALYSIS OF POISONING BY CARBON BISULPHIDE.

THE notes of the following cases were taken by my clinical clerk, Mr. P. de C. Potter:—

CASE I.—J. N., aged 24 years, was admitted to the Manchester Royal Infirmary on May 5, 1886, under the care of Dr. Ross.

Previous History.—The patient is an unmarried man, and never suffered from any serious disease up to the date of his present illness. He has always been well clothed and fed, has never indulged in any alcoholic excesses, and has for a considerable time before admission been a total abstainer. He has never been infected with syphilis. The patient is of Irish extraction, but has lived in Salford for the last eleven years. During the greater part of the time he has been in this country he was employed in a calico printing works; but being out of work about eight months ago, he obtained a temporary engagement in an india-rubber factory. In his new situation he was employed in the "curing-room," where he inhaled the fumes given off from certain chemicals used in the process of manufacturing, and said to consist chiefly of bisulphide of carbon. After working for a few weeks in this place the patient suffered from a burning sensation in the hands and face, and these were also hot to the touch and of a red colour. He found, however, that when he put his hands in cold water they immediately turned of a livid colour, and became cold and numb as if they were dead, or, as the patient at another time expressed it, "they looked just as if they had frost bitten." In consequence of the effect cold water had upon his hands, and to a less extent upon his face, he was obliged to wash himself in warm water. On getting home at night he suffered from a fidgety and restless feeling which prevented him from being able to sit still for more than a few moments at a time. After working for a few weeks longer the patient experienced tingling sensations and numbness in his feet and hands, his legs began to feel heavy and feeble, and he noticed a considerable degree of weakness at both wrists. He continued, however, at his work for some weeks longer, but owing to the progressively increasing weakness of his limbs he was at last obliged to desist. The patient now rested for two or three weeks, and during this time made a rapid and, he believes, a complete recovery, and then returned to his old work in the "curing-room."

He was, however, not many weeks at work before he felt the old uncomfortable sensation in the hands and feet, while the weakness soon became much greater than it had previously been. He now experienced the greatest difficulty in walking, and could scarcely hold anything in his hands, which, besides being feeble, trembled a good deal, more especially when he attempted to grasp anything.

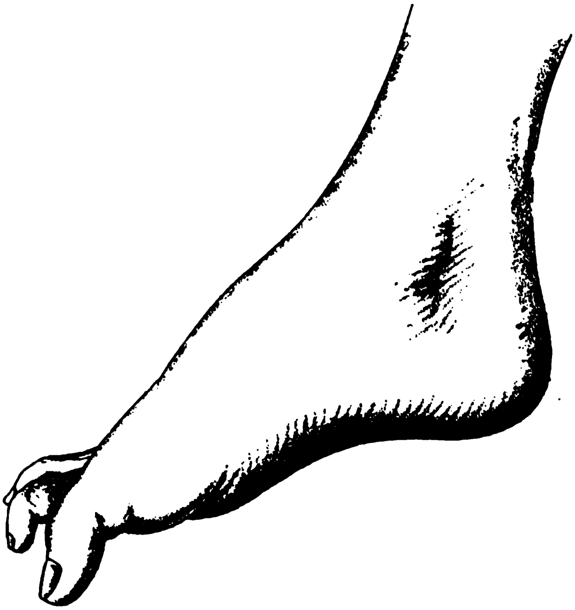
The senses of sight and hearing remained unaffected, but everything seemed to smell of the vapours of the factory even when he was away from his work, and his food seemed either to be tasteless or to taste only of the gas. The patient loathed the sight of food; he lost a stone in weight, and observed that the wasting of his legs and arms was out of all proportion to that of the rest of his body. On leaving his work in the evening he often walked like a drunken person, and talked a great deal of nonsense. He had at all times a stupid feeling, and his memory failed almost completely, while at night he was restless, and his sleep was disturbed by horrid dreams. In the morning he felt thoroughly miserable and depressed, and was glad to get back to his work, as inhaling the gas brought some relief, at first at least, to his feeling of wretchedness. The patient at last got so feeble that he could scarcely walk at all, and for the last four weeks he ceased to go to his work. He however, got a temporary engagement at a tarpaulin manufactory, but he soon found that he was unable to do the work owing to the weakness of his hands.

Present Condition.—The patient is a tall and fairly well nourished man, but the muscular masses of the extremities are considerably wasted, the emaciation being specially marked in the muscles of the legs and those of the forearms. When the forearms are held out horizontally in the prone position the patient experiences considerable difficulty in maintaining the hands extended on the forearms and the fingers at the metacarpo-phalangeal articulations, while the slightest pressure on the backs of the hands overcomes the contractions of the extensors and causes flexion at the wrist. When he grasps any object the hand becomes bent forwards on the forearm, and the greater the effort the patient makes to compress the object the more pronounced does the flexion at the wrist become, thus showing a predominance in the strength of the flexors over that of the extensors. The triceps muscle is flabby and wasted, and only acts feebly in extending the forearm against resistance. The supinator longus, the flexors of the arm, and the shoulder muscles are comparatively unaffected.

The patient can only extend the small toes very feebly at the metatarso-phalangeal joints, and when he is sitting on a chair with his feet flat on the ground he is unable to raise the ball of either foot. When sitting on a table with the legs pendulous the toes drop so that the backs of the feet are almost in a line with the anterior surfaces of the legs, or only form very obtuse angles with them. On being seated on a chair the patient can with considerable effort extend the leg on the thigh and raise the heel from the ground, but the slightest pressure on the leg, the thigh being supported, causes flexion at the knee-joint. The patient can draw the knees together with considerable force by the action of the adductors of the thighs, but separation of them is only effected in a feeble manner by the abductors. Flexion of the leg on the thigh is performed with great power, but the patient experiences considerable difficulty in attaining the erect posture, having to assist himself with his arms either by holding some article of furniture, or by grasping his thighs and pushing up the trunk as is done in pseudo-hypertrophic paralysis, thus showing that the extensors of the body upon the thighs are feeble.

On standing erect the patient maintains his feet about fourteen inches apart in order to widen his base, and on being got to place them side by side along their inner borders he sways slightly from side to side, and these swaying movements become greater when the eyes are closed, but he can still maintain the erect position, although with manifest effort.

The gait of the patient is the one which has been compared by Charcot to that of a high-stepping horse, and by Schulz to that of a dancing master. The chief peculiarity of this gait is caused by the fact that the muscles which produce dorsal flexion of the foot are paralysed. Let us suppose that the patient has advanced the right foot and planted it firmly on the ground. The abductors of the right thigh now contract, and the line of gravity is transferred to that side, so that it passes through the arch of the right foot, which is now the *active* one. The weight of the body having been removed from the left limb, which is now the *passive* one, the heel is slightly elevated, so that the toes alone rest on the ground, while there is the slightest possible flexion at the knee-joint, and a still less at the hip-joint. In ordinary locomotion a slight dorsal flexion of the foot would now be produced, the toes of the passive foot would thus be raised from the ground and the limb would swing forwards by its own weight without muscular action. The patient, however, is unable to produce dorsal flexion of the foot, and, consequently, compensatory movements have to be effected in order to clear the toes off the ground. These movements consist of an unusual degree of flexion of the thigh upon the trunk, and of the leg upon the thigh, which causes a shortening in the length of the whole limb. The heel of the passive foot becomes raised from the ground in direct proportion to the elevation of the knee-joint, by the flexion of the thigh on the body, but, owing to the paralysis of the anterior muscles of the foot, the toes continue to drop until their further depression is arrested by the anterior ligaments of the ankle-joint. The consequence of the continued dropping of the toes while the heel is being elevated, is that an observer, standing behind the patient, sees more of the sole of the foot at each forward step than in ordinary locomotion, and on standing laterally, notices the drop of the toes and the unusual elevation of the knee with each advancing step.



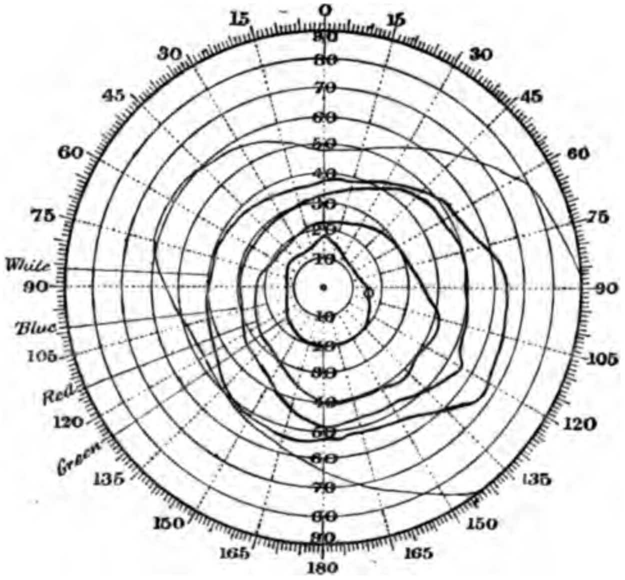
When the patient is laid in bed the feet assume the position of a double ankle drop. The small toes are hyperextended to a very slight degree at the metatarso-phalangeal joints, and flexed at the phalangeal joints, but the big toe is flexed at both the metatarso-phalangeal and the phalangeal joints. The annexed drawing, kindly taken by my clinical clerk, Mr. Marshall, from a case of alcoholic paralysis, accurately represents the position of the toes and foot in this case, with the single exception that the small toes were not quite so much hyperextended as they are represented in the figure. The patient can voluntarily extend, to a slight degree, the small toes at the metatarso-phalangeal joints, and can produce a little eversion of the foot, but he is quite unable to produce dorsal flexion at the ankle joint, extension of the big toe, or inversion of the foot on either side. These observations show that a slight degree of motor power remains in the long extensors of the toes and the peroneal groupe, but that the extensors of the big toe and the tibialis anticus are completely paralysed. The consequence of this distribution of the paralysis is that the sole of the foot is not only directed backwards towards the plane of the bed, owing to paralysis of the muscles causing dorsal flexion of the foot, but that it also has a slight inclination outwards, or is somewhat everted, because the peroneal muscles have still retained some degree of motor power, while the tibialis anticus is completely paralysed.

The paralysed nerves and muscles react to a moderate strength of the faradic current, and, indeed, it can hardly be said that the faradic contractility is even lowered. The extensors of the toes on the right side give a minimum contraction on cathodal closure to 20 cells and on anodal closure to 15 cells, and on the left on both cathodal and anodal closure to 20 cells Leclanché. In the extensors of the thigh and those of the forearm anodal closing contraction is equal to or greater than cathodal closing contraction. The reflex of the sole is absent, but the other cutaneous reflexes are normal. The patellar tendon reactions are absent.

The patient complains of a feeling of numbness and tingling in his toes and feet and in the tips of his fingers, but the sensations of pain, touch, and temperature are found to be normal, or nearly so, to objective examination. Pinching of the skin over the external aspects of the legs appears to cause an unusual degree of pain, but there is no undue sensitiveness of the muscles on palpation. The patient complains that he has no proper taste for his food, but he readily identifies salt and sugar when placed on his tongue. He also says that his smell is blunted, but he recognises camphor when a piece is applied to his nostrils. The sense of hearing is normal.

May 27.—The condition of vision was examined by Dr. Little this morning, and he reports as follows: "The patient does not complain of any defect of vision. The acuteness of vision is normal in both eyes, and the fundi are apparently healthy. The perimetric chart, on p. 188, of the field of vision for the right eye shows a considerable restriction for all colours. A general examination of the left eye shows that it is similarly affected." In the chart the dark lines represent vision for white, blue, red, green, in their order, from without inwards.

Beyond the muscular wasting, no *trophic* changes are noted. The hands, when warm, are reddish, and are moist and clammy to the touch, but when placed in cold water they immediately assume a livid colour, and become cold, looking just as if they had been frost-bitten, or in the early stage of Reynaud's symmetrical gangrene. The patient lost all sexual desire a few weeks after he began work in the curing-room, and even at the present time he never has any erections. The loss of this function was not preceded by a stage of sexual excitement. No marked psychical symptoms have been observed, although the patient complains very much of loss of memory.



The treatment adopted consisted of rest, good nourishment, massage, and galvanism. A tonic mixture was prescribed, with the view of aiding digestion.

During a residence of a month in the Royal Infirmary the patient improved considerably, but he was still unable to produce dorsal flexion of the feet, and the patellar tendon reactions remained absent. He was now sent to the Convalescent Hospital at Cheadle, and six weeks afterwards he appeared as an out-patient at the Infirmary, when scarcely a trace of the previous paralysis could be detected. The patellar tendon reactions had reappeared, but were still sluggish, and dorsal flexions of the feet, although capable of being effected, were not produced with normal strength.

The patient soon afterwards disappeared from observation. He was, however, again examined by Dr. Little,* in May, 1887, and the fields in both eyes were then found quite normal.

CASE II.—P. S., aged 36, was admitted to the Manchester Royal Infirmary, under the care of Dr. Ross, on May 12th, 1886.

Previous History.—The patient is of Irish extraction, but he has lived in Pendleton for the last twenty-four years. He is a moulder, and was always engaged at his own trade until work was scarce at the beginning of last winter, and up to that time he enjoyed good health, never being laid up for a day with any kind of illness. The patient had always been well fed and clothed, and although he is not a total abstainer, he has never indulged in alcoholic excess, and was not a heavy smoker. He has been married eight years, and has one child, who is living and healthy. His wife never had miscarriages or still-born children, and there is nothing in his history pointing to syphilis. About nine months ago, being out of work, he obtained employment in an india-rubber factory, and worked in the "curing-room," where he was exposed to fumes supposed to be bisulphide of carbon gas. He worked for three months in this place before he felt any ill effects from the gas, but at the end of this time his appetite completely failed, his food tasted of the gas, and everything about him seemed to smell of it. He soon found himself unable to read, the letters seeming to run into one another. He also became deaf on the left ear, but he thinks his right was not at any time much affected. His lower limbs now felt heavy, and he became so feeble in a short time that he could scarcely walk, and occasionally fell down. About the same time he experienced tingling sensations and numbness of his hands; they also began to tremble, and his grasp was so weak that he could scarcely hold anything. He also experienced similar tingling sensations in the toes, and his feet felt so numb and dead that it seemed to him as if he were treading on something soft instead of on solid ground, and walking, at all times somewhat uncertain, became specially insecure in the dark or on closing his eyes. His sexual appetite failed entirely after working for a few weeks in the "curing-room," and this loss was not preceded by a stage of undue excitement. On putting his hands in cold water they immediately became numb and white as if they were frost-bitten.

It is difficult to ascertain from the patient's description whether he suffered from genuine cramp in the legs or from pains shooting along the course of the nerves, or from both of these disorders. He suffered at least from some kind of pains in the lower extremities which caused him on going to bed to wish for a frequent change of the position of his legs, and greatly disturbed his rest, while the snatches of sleep he obtained were disturbed by horrid dreams, in which he fancied himself surrounded by cats and other animals. In the morning he got up drowsy and unrefreshed, and suffering from an intense headache, chiefly limited to a spot in the forehead and bteween the eyes, which lasted with more or less intensity throughout most of the day. He was, indeed, so utterly miserable and mentally depressed on getting up that he wished himself dead, and at the same time had a longing to get back to his work, as he found by experience that the inhalation of the fumes would bring temporary relief to his sufferings. After breathing the gas for a short time his mental depression gave place to a joyous feeling which was but short lived, being replaced in the afternoon by an indescribable feeling of apathy and wretchedness. The patient's memory failed very much, his recollection of recent events being specially defective.

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* Little (Dr. D.). "Tonic amblyopia—bisulphide of carbon."—*Ophthalmological Transactions*, Vol. VII., London, 1887, p. 74.

He sometimes found himself talking nonsense while at his work, and he occasionally fancied that he was surrounded by cats, but he does not think that in this respect he was affected to anything like the same degree as some of his fellow workmen. He says that some of the workmen when under the effects of the gas become very loquacious, and at other times talk a great deal of nonsense. One man on coming to his work in the morning told his comrades that he was in Liverpool the previous night, a statement which could not possibly have been true. Another workman, apparently to escape from some imaginary danger, jumped through a window, ran across an open court, and having crept under a joiner's bench, tried to hide by covering himself with shavings. One or two of the workmen have gone quite mad, and have been sent to a lunatic asylum.

Present Condition.—When the patient is seated on a chair, with his feet flat on the ground, he is unable to raise his toes off the ground by dorsal flexion of the feet. He has sufficient strength to extend the leg on the thigh, and to raise the heel off the ground, but the slightest pressure on the front of the leg suffices to cause flexion at the knee-joint when the thigh is supported. When the forearms are held out horizontally, the hands being in the prone position, the patient can, with much effort, maintain the hands extended upon the forearms and the fingers extended upon the hands, but the slightest pressure on the backs of the hands causes flexion at the wrists, and on the backs of the fingers flexion at the metacarpo-phalangeal joints. When the patient is laid on his back in bed there is double ankle drop; the big toe is flexed into the sole at both joints, but the small toes are slightly hyperextended at the metatarsal-phalangeal and flexed at the phalangeal joints. It is, indeed, unnecessary to describe in detail the distribution of the paralysis in the case of P. S., inasmuch as it corresponds in almost every particular to that observed in the case of J. N., just reported, the gait of the former being also so like that of the latter, as to make a separate description superfluous. When the patient stands with his feet approximated, and with closed eyes, he sways from side to side and maintains the erect posture with manifest effort.

The following reactions were obtained in the affected nerves and muscles to electrical examination:—The faradic irritability was diminished in the extensors of the forearm, and in those of the legs and thighs, as well as in the nerves which supply these muscles, but all the nerves and muscles reacted to a moderately strong current.

The following reactions to the galvanic current were obtained:—

Extensors of left forearm—	KSc. = 20	ASc. = 15	Cells Leclanché.
" "	KOc. = 40	AOc. = 30	" "
Extensors of right forearm—	KSc. = 15	ASc. = 15	" "
" "	KOc. = 30	AOc. = 30	" "
Extensors of toes: left—	KSc. = 20	ASc. = 10	" "
" "	KOc. = 45	AOc. = 35	" "
Extensors of toes: right—	KSc. = 20	A.Sc. = 20	" "
" "	KOc. = 35	AOc. = 30	" "

The patient complained of numbness and tingling of the hands and feet, and there was a slight diminution of the sense of pain, as tested by pricking and of touch, as tested by several points, in the outer aspects of both legs and feet, but the sense of temperature did not appear to be affected. The various forms of cutaneous sensibility were found to be normal in the remaining parts of the body. Moderate compression of the affected muscles did not cause pain. On being asked to touch, with closed eyes, his nose, with the tip of his index finger, he touched somewhat wide of the mark at first, but effected the movement accurately with a little practice. The reflex of the sole was absent, but the cremasteric and other cutaneous reflexes appeared to be normal. The patellar-tendon reactions were absent.

The patient is able to distinguish salt, sugar, and quinine, when placed upon the tongue, and he also recognises camphor when applied to his nostrils, but he says that both of these senses are much blunted, and that everything seems to taste or smell of the gas. He hears the ticking of a watch on the right side when it is two feet from the ear, but not on the left side until contact is made. There is no undue collection of wax in the external meatus, and no discoverable disease in the tympanic membrane or external ear.

During the patient's residence in the Infirmary he improved considerably. The patellar-tendon reactions reappeared, but were still sluggish. The extensors of the forearms acquired a considerable degree of motor power, but the muscles which produce dorsal flexion of the feet still remained comparatively paralysed, and the gait of the patient presents a high-stepping action which is so characteristic of paralysis of these muscles. The treatment prescribed was the same as in the case of J. N.

The pupils are dilated, but contract readily to light and to accommodation. The patient says that he cannot read because all the letters run into one another. His colour vision is defective; purple he calls white, and is unable to distinguish red from blue.

The patient's hands look as if they were frost-bitten, when dipped in cold water, but besides the wasting of the muscles there are no trophic disorders. No decided psychical disturbances are apparent, although the patient is morose and discontented, and says that his memory is still very defective.

May 27th. The patient was sent to Dr. Little to-day for examination, and the following report of the state of his vision was obtained :—

"Sight failing for about six months. Vision of right eye is equal to 18, and of left to 16 Jaeger. Refraction shows a slight degree of hypermetropia. An ophthalmoscopic examination showed that the right optic disc is distinctly pale but clear, while the left is pale with a faint haze at some points in the margin. The fundus of each eye is healthy in other respects. The field of vision is much contracted in each eye for white and blue, while red and green are absent. The annexed perimetric tracing represents the field of vision for the different colours in the right eye. (See page 193.)

After a month's residence in the Infirmary the patient was discharged improved. Dr. Little,[*] who examined him again in June, 1887, gives the following report of his condition :—"He had almost completely recovered his health and sight. Vision in each eye was equal to $\frac{20}{30}$, the optic discs were pale but clean, the large retinal vessels were narrow, perception for all colours was normal, and the fields for all colours were also normal."

Having put these cases of paralysis from the inhalation of bisulphide of carbon on record, it is not my intention to enter, at present at least, upon a general consideration of the subject. It will suffice to say that this form of paralysis belongs to the group which is caused by various toxic agents, such as alcohol, lead, arsenic, and certain animal poisons like that of diphtheria. The tendency of pathologists at present is to regard all the diseases comprised in this group as being caused by a neuritis of the peripheral nerves, and the group has consequently been named progressive multiple neuritis, or *polyneuritis multiplex.* From the resemblance which several of the symptoms of these cases—such as the sensory disorders, the absence of the patellar tendon reactions, and the gait—bear to those observed in locomotor ataxia, these diseases have also been named *pseudo-tabes*, a term which is badly chosen, and ought on every ground to be rejected. Locomotor ataxia is only exceptionally attended by any paralysis, and the high-stepping gait of progressive multiple neuritis could only be mistaken for the disorderly and thumping gait of ataxia by very careless observers.

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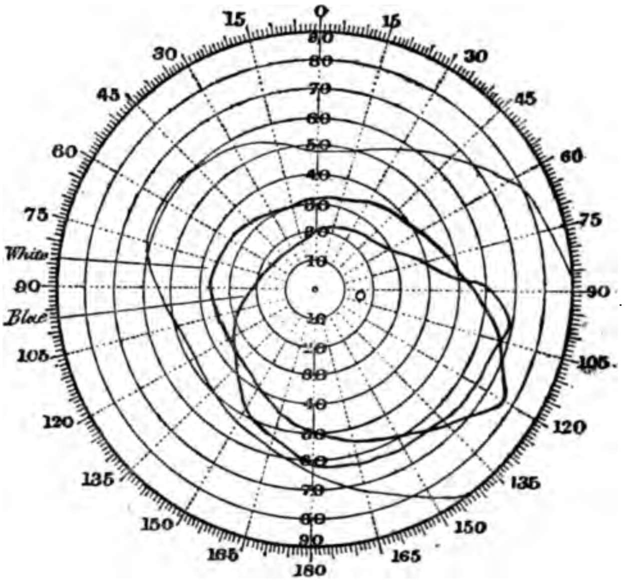
[*] Little (Dr. D.). Loc. cit., p. 74.

The paralysis which results from the prolonged inhalation of bisulphide of carbon resembles alcoholic paralysis more than it does any other form of progressive multiple neuritis. One of the most characteristic features of alcoholic paralysis is, however, an intense hyperæsthesia of the affected muscles, which makes the slightest pressure of them almost insupportable to the patient, and this symptom was entirely absent in the cases just described. The amblyopia and loss of colour vision, which was present in a marked degree in Case II., is absent in alcoholic paralysis, unless it be complicated by tobacco amblyopia.



Inhalation of the fumes of bisulphide of carbon sometimes gives rise to acute symptoms closely resembling those of acute alcoholism. The similarity of the symptoms in the workman described by P. S. as jumping through the window and hiding under the shavings to the symptoms of delirium tremens, is unmistakable. It also gives rise occasionally to various hysterical symptoms.*

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* Marie (P.). "Sulfure de carbone et hystérie."—*Bull. et Mém. de la Soc. Méd des Hôp. de Paris*, 1888.

The symptoms in the cases just recorded are so typical of the effects produced by exposure to the fumes of bisulphide of carbon in india-rubber factories that no useful purpose will be served by entering upon a detailed analysis of the cases reported by other authors, but a full reference to the literature of the subject is appended.

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(*d*) THE PARALYSIS OF POISONING BY NAPHTHA.

The notes of the following case were taken by my clinical clerk, Mr. H. Part.

N. B., aged 44 years, entered the Royal Infirmary under the care of Dr. Ross on January 8th, 1890.

The patient states that he has always been a very strong and healthy man up till about three or four years ago, when he had an attack of inflammation of the lungs, and was then an in-patient in the Infirmary, under Dr. Morgan. He says that he has not been quite free from pain in the side since that time. For the last eight years he has worked in an india-rubber factory, and he has been much exposed to the smell of naphtha, and the nature of his work obliged him to immerse his hands and arms frequently in pure naphtha. He was also exposed to the fumes of bi-sulphide of carbon, but he did not work in the "curing room," and he attributes his symptoms to the naphtha and not to the carbon compound. The patient admits that he drank heavily at one time, but has been very abstemious in this respect for the last six months. He has for a long time suffered from numbness and coldness of the hands and feet, and been troubled from time to time by cramps of the calves of the legs and thighs on going to bed. He has occasionally had attacks of retching and sickness in the morning, which were especially severe after he had been drinking freely. The patient says he has felt for a long time very drowsy whilst at work, and on getting home he would fall asleep by the fire, but on getting to bed he became very wakeful again, and the little sleep he got during the night was disturbed by horrid dreams. He felt very miserable and depressed in spirits every morning, but after being at work for two or three hours his spirits revived and he felt comfortable and happy until the afternoon, when he became very drowsy and often fell asleep whilst standing at work. As soon as he got home he fell asleep in his chair, but only to wake soon and to find himself quite unable to sleep at night. For the last two or three months he has been troubled when in bed at night with seeing images and visions. These took at first the form of stars and balls of fire floating in a halo of light, but after a time he began to see the faces and figures of persons he knew or read about. He saw, for example, "Jack the Ripper," and he now gives a very minute and vivid description of his appearance. At a later period he began to see rats running about his bed, and on one occasion he counted 60 of them in a row. He has also been exceedingly irritable and quarrelsome at home, and nobody could do anything to please him. He got suspicious of his wife and thought she would do nothing to help him. On one occasion he struck her, and he would have killed her many a time if he "got the chance." His family history is good, and no constitutional taint of any kind can be discovered. About a month ago he was seized with a troublesome cough and a sharp cutting pain in his left side, which was worse on breathing and on exertion, and was very severe during attacks of coughing. After trying various household remedies, without effect, he became an out-patient at the Infirmary, and was subsequently admitted as an in-patient.

Present Condition.—The patient is a well-nourished and muscular man of medium height. He complains of a stitch in the left side of his chest, which is aggravated on deep inspiration. He is also troubled with a cough, and expectorates a considerable quantity of a thin frothy sputum. The chest is barrel-shaped, and hyper-resonant on percussion, but the difference in its circumference on a level with the nipples during maximum inspiration and maximum expiration is 2½ inches. On stethoscopic examination a few scattered rhonchi are heard all over both sides of the chest, and a friction sound is heard on the left side in mid-axillary line, about the level of the eighth rib. There is no dulness over the left side of the chest, and the vocal fremitus is not perceptibly diminished, as compared with corresponding parts of the opposite side. The heart is well covered by hyper-resonant lung, and the area of deep dulness is not easily made out, but it is not appreciably enlarged. The heart sounds are normal, and are free from murmur. A slight degree of capillary pulsation can be elicited on scratching the forehead. The pulse is 82 per minute, regular, and of fair strength. There is a slight evening elevation of temperature. The urine is acid, specific gravity 1,022, and free from sugar and albumen. The knee-jerks are absent, and although the patient can perform all the movements of the lower extremities as he lies in bed, they are found to be feeble when tested by apposing actions. His grasp is feeble, and he is unable to approximate the tips of the little fingers and thumbs without flexion of the distal phalanges of the thumbs. There are no manifest distortions of the feet or big toes, and the gait, although feeble and uncertain, is not high-stepping. The patient complains of some numbness of the fingers and toes, and the calves of the legs are now tender to pressure, but there are no sensory disorders to objective examination. The plantar and cremasteric reflexes are sluggish, but the abdominal and epigastric reflexes are normal. The pupils are somewhat dilated, but react to light and accommodation, and there are no disorders of the ocular movements. There is no restriction of the fields of vision; colour vision is good, and the optic discs are found to be normal on ophthalmoscopic examination.

Jan. 25.—Since admission the patient has slept well at night, and his cough has nearly ceased, while the pain in his side has all but disappeared. The patellar tendon reactions are still absent. He was now discharged.

In this case the paralysis was not very pronounced, but taking the general weakness of the extremities, the inability to perform accurately special movements of the fingers, the loss of the patellar tendon reactions, and, above all, the characteristic psychical disorders, there can be no question that the patient was suffering from some form of peripheral neuritis. At the same time, the confession of the patient, that he was in the habit of taking large quantities of alcoholic stimulants, renders it difficult to determine how much of the symptoms is to be attributed to alcohol and how much to the poisons to which he was exposed while at his work. That the latter poisons, however, were chiefly to blame, was made highly probable by the statements of a fellow-workman of N. B., who, by a singular coincidence, happened to occupy the next bed to him. The second patient was a young man of about twenty years of age, of fairly healthy appearance, who was suffering from a slight attack of bronchitis. He had also numbness of the extremities, absence of the patellar tendon reactions, and other indications of a slight degree of multiple neuritis, but I regret to say that the notes of his case have been mislaid. On listening to N. B., as he was describing his miserable and sleepy condition while at work, the second patient said: "I worked in the same room with this man, and that is exactly how I used to feel." He then described to us how depressed and miserable he felt in the morning, and how, as he got to his work, he soon became comfortable and happy, until the afternoon, when he became drowsy and apathetic. He also was sleepless at night, but his hallucinations never got beyond the stage of seeing balls of fire, and occasionally faces. The statements of the second patient possess all the more value because he was a total abstainer from alcohol. Supposing, then, we assume, that the symptoms in these two cases were caused not by alcohol, but by the poisons to which they were exposed while at work, now comes the question as to whether the naphtha or the bi-sulphide of carbon is to blame. These men were unanimous in attributing their symptoms to naphtha. They were quite aware of the dangers of bi-sulphide of carbon, but they said that they were not much exposed to it, as they did not work in the 'curing room."

(e) THE PARALYSIS OF POISONING BY DI-NITRO-BENZOLE.

A NEW explosive compound, introduced under the name of "roburite," has recently been used for blasting purposes in mines, and essentially the same agent appears to be sold under the name of "sicherheit."[*] According to Professor Dixon,[†] roburite is an intimate mixture of di-nitro-benzene, chloro-nitro-benzene, and ammonium nitrate. It was soon discovered that the workmen engaged in handling and exploding it suffered from great weakness and a remarkable deterioration of the general health.[‡] I am indebted for an opportunity of examining several cases of poisoning by this agent to Mr. Berry, surgeon, of Pendlebury, and for the notes of the cases to Mr. A. E. Berry, at that time acting as one of my clinical clerks.

Case 1.—G. G., aged 42 years, sought Mr. Berry's advice on February 28th, 1889. He has worked in the same mine for many years, and has always enjoyed excellent health up to the commencement of his present illness. He has always been a temperate man, and has been, of late, a total abstainer. About five months ago he began to use "roburite," and soon afterwards his eyes and face felt to "smart." He next felt a pain under his right shoulder blade, and he suffered from severe headaches, which were at times situate in the forehead and temples, and at other times in the back of his head. Two months after he had begun the use of this agent he complained of numbness of his finger tips and of his toes, and when he sat down his fingers and toes "would go to sleep." Soon afterwards he began to suffer from shortness of breath and attacks of palpitation, these symptoms being especially troublesome when he exerted himself, as in ascending a hill or a stair. During the last few weeks he has suffered from shooting pains in the lower extremities, which were accompanied by muscular twitchings; he lost all "spring" in walking, his grasp became feeble, and his legs felt heavy and weak. He also felt drowsy and apathetic, and his eyes felt so weak that he had to "blink" on entering a lighted room, although the acuteness of vision or his perception of colour did not seem to be impaired. He found that he had lost about 14lbs. in weight. His urine was so high coloured that his wife several times remarked that she thought he must have been passing blood. The patient states that he has never handled the powder.

Present Condition.—The patient is a strong well-built man, but his face looks intensely pallid, while his lips are of a bright blue tint, and quite different from the dusky blue colour met with in mitral disease and chronic bronchitis. The conjunctivæ have a slight icteric tint. The patient complains of drowsiness, general feebleness, and a great indisposition to exert himself. His fingers and toes feel numb, and at times "go to sleep," while on the slightest exposure his fingers become white, bloodless, and cold, and feel as if they were dead. He has consequently a great dread of touching any cold object, and always washes in warm water. He also suffers greatly from cold feet. There are no marked disorders of cutaneous sensibility to objective examination. The patient's grasp is much feebler than that of a healthy man, especially than that of a man habituated to heavy manual work. The spaces between the metacarpal bones are distinctly grooved over the back of the hands, and the abductor indicis, and the muscles of the thenar and hypothenar eminences in both hands are soft, flabby, and distinctly wasted. The patient cannot, without considerable effort, bring the tip of the thumb of the left hand into contact with the tip of the index finger, and still greater difficulty is experienced in bringing the tip of the thumb into contact with the tip of the little finger. He is also unable to flex the thumb strongly into the palm without flexing it at the phalangeal joint. The power of adducting the thumb is also feeble. The movements of the right hand are similarly affected, but all the tests could not be applied, because of the loss of his right index finger.

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[*] Sykes (W.) and Twigg (F Graham). "Case of poisoning by new 'sicherheit' explosive."—*The Lancet*, Vol. II., 1889, p. 127; and Knight (Henry John), "Poisoning by 'sicherheit' explosive."—*The Lancet*, Vol. II., 1889, p. 244.

[†] See *The Lancet*, Vol. I., 1889, p. 1100.

[‡] See White (R. Prosser). "The toxic effects of nitrobenzole."—*The Practitioner*, Vol. XLII. London, 1889, p. 14.

The patient seems to have lost all elasticity and spring in walking, but the toe of the advancing foot does not manifestly drop. When the patient sits on a chair, and places his uncovered feet flat on the ground, he is able to produce dorsal flexion so as to raise the toes and ball of either foot from the ground, and when the whole foot is raised from the ground there is no distinct ankle-drop. The big toe of the right foot, however, is slightly flexed into the sole at both the metatarso-phalangeal and phalangeal joint, and the patient experiences greater difficulty in raising it from the ground than he does with the corresponding toe of the left foot. The patient still complains of twitchings of the lower extremities, which occur chiefly when he is laid in bed at night. The patellar-tendon reactions are absent on both sides. The reflex of the sole is normal, but the cremastric reflex appears to be absent, and the patient states that for the last month he has lost all sexual desire. The special senses are not perceptibly affected to objective examination. The field of vision is not restricted on either side, and the perception of colour is good. The patient, however, states that he is subject to attacks of total blindness, which come on when he directs his eyes upwards. He states that these attacks usually come on when he combs his hair. The mirror in which he looks is hung above the level of his eyes, and when he is engaged in combing a film comes over his eyes, and he is soon totally blind; he now bends his head forward and downwards, and his sight almost immediately returns.

The urine is high coloured, and, on cooling, deposits a large quantity of urates, the sediment being of a dark pink colour. Traces of biliary colouring matter are found in it by means of the usual tests.

On the blood being examined by means of the spectroscope, it was found that the two bands to the violet side of D line were not reduced to one, on the addition of ammonium sulphide, so quickly as in the blood of a healthy man, obtained for comparison.

April 3.—G. G. visited the Infirmary to-day. He is considerably improved in general health. The patellar-tendon reactions are still absent, and there are slight indications of paralysis of the small muscles of the hands and feet, but his face has lost much of its pallor, and the blue tint has nearly disappeared from his lips. He expresses himself as feeling much stronger and better in health than he was when last examined.

Case 2.—E. S., aged 51 years, came under Mr. Berry's care at the beginning of March, 1889. The following report was taken March 4th: The patient always enjoyed excellent health until he began the use of "roburite." About four months ago he worked for three weeks in a mine where "roburite" was used, but he only suffered at that time from some headache. Three weeks before he came under observation he began to use "roburite" again, and after being at work for nine days he complained of dizziness, and felt feeble in his lower extremities. He now felt a constriction across the chest, and suffered from palpitation and shortness of breath on exertion. His face felt to smart a few days after he began to use this agent, and soon he suffered from griping pains in the abdomen, and these were followed by loathing of food, nausea, and vomiting. He complained of numbness and tingling of his finger tips, noises in the ears, and dimness of vision, with dark specks floating before his eyes. At the end of three weeks from beginning to work with "roburite," he suffered very much from nausea and vomiting, and felt so weak that he could scarcely walk home. He now desisted from work, but, although the vomiting ceased, he felt drowsy and apathetic still, and his lower extremities seemed to become gradually weaker, so that now he felt unable to walk more than a few hundred yards on level ground without feeling greatly fatigued. The patient states that he never handled the powder. In opening the cartridges some of the powder might get on the fingers, but he was always careful to wash his hands immediately.

Present Condition.—The patient is strongly built, but he looks ill and feeble. His face is very pallid, and his lips have a bright blue colour. A soft systolic murmur, having its maximum intensity over the pulmonary area, and a continuous humming murmur over the jugulars in the neck are audible. He complains of great drowsiness and general weakness. His fingers tingle, and become pale and numb on the slightest exposure to cold. The grasp of either hand is very feeble, and he experiences some difficulty in bringing the point of the thumb into apposition with the tips of the other fingers, and in performing other special movements. The patient has lost all spring in walking, and has difficulty in standing on his toes, but there is no ankle drop and no flexion of either of the big toes into the sole either at the phalangeal or metatarso-phalangeal joints. The patellar-tendon reaction is lively, probably exaggerated, on the right, and elicited with difficulty on the left side. All the muscles react to a moderate faradic current, but the electric sensibility is diminished on the whole of the right as compared with the left half of the body. There are no other disorders of sensibility to objective examination, and no affection of the special senses. The urine is high coloured, and contains a large sediment of dark-coloured urates and biliary pigment, but no hæmoglobin.

March 18.—The patient has gradually improved in health, and all the symptoms are now less marked than they were when he was first examined.

Case 3.—E. J., aged 46 years, came under observation on February 28th, 1889, when the following notes were taken. The patient is a powerfully built man, and he states that he has never suffered from any illness until about four or five months ago, when he began to use "roburite." Three weeks after he began the use of this agent, he suffered from headache, throbbing in character, and situate in the forehead and temples. He soon suffered from shortness of breath on exertion, and palpitation, but it is not until about a month ago that he, himself, and his friends noticed the pallor of his face, and the blue colour of his lips. He now lost his appetite, and suffered from constant nausea and occasional attacks of vomiting, and his sight had become dim. He does not seem to have noticed much numbness and tingling of his finger-tips, but he had shooting pains passing down the outside of his legs, and, at times, his lower extremities became suddenly so weak that he had to desist from work for some hours, and a few days ago, the weakness became so great and persistent that he left off work altogether. He states emphatically that he never handled the powder. The cartridge has, however, to be opened, and it is hardly possible to avoid getting some of it on the hands.

Present Condition.—The patient's face is intensely pallid, and his lips are of a bright blue colour, but he is not emaciated, and he has all the appearances of having been a powerful man. He complains of apathy, great drowsiness, loss of memory, and a feeling of great weakness. A humming murmur is heard over the large veins in the neck, and a soft systolic murmur is audible over the heart, having its maximum intensity in the pulmonary area. The patient's hands are moist and clammy, and become very cold on the slightest exposure. The spaces between the metacarpal bones are slightly grooved on the backs of the hands, but the thenar and hypothenar eminences are not perceptibly wasted, although they feel soft and flabby when handled. The patient has some difficulty in performing the special movements generally used to test the strength of the small muscles of the hand. The extensors of the fingers and wrist do not appear to be relatively weaker than the flexors of the forearm, although both groups of muscles are very feeble, especially when it is considered that the patient has been inured to heavy manual labour. The patient complains of great weakness of his lower extremities, but there is no manifest ankle-drop, and there is no distortion of the toes. The patellar-tendon reactions are very lively on both sides. There is a slight degree of comparative anæsthesia of the whole of the right half of the body, but about three inches below the inferior angle of the scapula of that side there is a patch of skin which is much more sensitive to the prick of a pin than the skin of the opposite side. The urine is high coloured, and deposits a quantity of dark-pink urates on cooling, but it does not give the reaction of blood to the guaiacum test.

March 16.—The patient is still anæmic, and complains of drowsiness, frontal headache, palpitation, and shortness of breath on exertion, but his appetite has improved, and he feels stronger than he did.

Case 4.—L. J., aged 28 years, came under the care of Mr. Berry about the end of February. The patient states that he had always enjoyed good health up to about four months ago, when he began the use of "roburite." Soon after he began to use this agent his eyes and face felt to smart, but he disregarded this feeling, and it is only about a month ago that he became convinced that his health was being seriously impaired by it. He now complained of tightness across the chest, and difficulty of breathing on the slightest exertion, and soon after he suffered from numbness and tingling of the fingers and toes, and from shooting pains in his lower extremities. The patient now became progressively weaker, until at last he could scarcely walk home, and was obliged to desist from work. The patient never handled the powder, and never even opened a cartridge.

Present Condition.—The patient is a well-built and muscular man, but his face is very pale, and his lips are of a bright blue colour. A soft systolic murmur is heard over the heart, and a continuous humming sound over the large veins of the neck. His hands and fingers are numb and feel to tingle, and he suffers much, and for the first time in his life, from cold feet. His grasp is fairly good, but much feebler than the grasp of one habituated to heavy manual work might be expected to be. The signs of wasting of the small muscles of the hand, and of a difficulty in performing special movements of the fingers are only present in a slight degree. The patient states that he has lost his spring in walking and in ascending a stair, but there are no definite signs of special paralysis of the muscles of the legs or of the small muscles of the feet. There are no sensory disturbances to objective examination. The patellar-tendon reactions are so lively that they must be regarded as being exaggerated. The patient finds that on the slightest exertion his body becomes bathed in a profuse perspiration. He feels drowsy, and disinclined to exert himself, but there is no appreciable loss of memory.

Case 5.—J. L., aged 28 years, came under observation about the end of February. He has always been a strong and healthy man until he commenced the use of "roburite" three months ago. Soon afterwards he felt his face to smart, and his eyes overflowed readily with tears, and he had to "blink" frequently when he entered a lighted room. Two months ago he began to suffer from pains in the back of the head and over the temples, and complained of noises in the ears and dimness of vision. He was much troubled with numbness and sensations of "pins and needles" in his fingers, and he found that when he rested his left arm for a short time on a table, the third and fourth fingers would become as if they were dead. For the last few weeks he was troubled with shortness of breath and palpitation on exertion, and at last he became so weak that he felt himself unable to work any longer. He felt a great disinclination to exert himself, and was so drowsy that he often fell asleep over his breakfast, and had to be aroused by his companions. The patient states that he never saw the powder, and he certainly never handled it.

Present Condition.—This patient is a well-built and muscular man, but he has the pale face and blue lips which is so characteristic of all these cases. The pulse beats 100 in the minute, and the action of the heart is excited, but no murmur is audible. He complains of numbness and tingling of his fingers and toes, but there are no sensory disorders to objective examination. His eyes are very sensitive to light, so that he knits his brows and half closes his eyelids when he stands opposite a window. The patient's grasp is by no means so strong as might be expected from one of his occupation, and he soon tires of walking, but there are no decided signs of special paralysis or wasting of the muscles of the hands or feet. The patellar-tendon reactions are normal. The patient feels drowsy and apathetic.

March 18th.—The patient presented himself for examination to-day. He is still pale and weak, but is much improved in health and general appearance since the date of the last report.

Case 6.—B. B., aged 45 years, a collier, states that he had always been a strong and healthy man until he began the use of "roburite" early in February this year.

The patient states that soon after he began the use of this agent he noticed that "the fumes caused his eyes and face to tingle." He soon suffered from headache, chiefly situate over the forehead and temples, and throbbing in character, noises in the ears, weakness of sight, and seeing stars before his eyes. About a fortnight after he began the use of this agent, and only one week before he desisted from work, he suffered very much from nausea, and vomited almost daily. He also complained of shortness of breath and palpitation on exertion, aching pains in his legs, and a feeling of great exhaustion and weakness on walking a short distance. The patient states that he opened the cartridges, but that very little of the powder ever touched his fingers.

Present Condition.—The patient complains of the usual numbness and tingling of his hands and fingers, and of great weakness, but the signs of a special paralysis of the muscles of the hands and feet are only slight, and there are no sensory disorders to objective examination. The patellar tendon reactions are very sluggish, and it is, indeed, doubtful if they are not absent.

March 18th.—This patient gained strength since last report, and there is a great improvement in his general appearance.

It is hardly necessary to point out that the numbness and tingling of the hands and feet, the local asphyxia of the fingers on exposure to cold, the muscular weakness, with difficulty in executing certain special movements, and the sluggishness or absence of the tendon-reactions present in these cases are the well-known indications of an early stage of multiple neuritis; and, indeed, the symptoms in these cases are so strikingly similar to those observed in the cases of carbon bi-sulphide poisoning, already reported, that it can hardly be doubted that the two affections own one common pathology. It may, however, be said that the poisonous agent was not di-nitro-benzol, but carbon monoxide, generated by imperfect combustion during explosion, and the observation made by Mr. A. E. Berry, in the case of G. G. (Case 1), that the two bands of the oxy-hæmoglobin were not so readily reduced by the addition of ammonium sulphide as the bands in healthy blood, lends countenance to this view. At the same time it must be remembered that di-nitro-benzol and its derivatives exercise a poisonous action on the hæmoglobin, and the bright blue tint of the lips of these men, which differed greatly from any form of lividity met with in imperfect aeration of the blood, conclusively shows that di-nitro-benzol or its derivatives had been absorbed into the blood, and we consequently see no reason to doubt that it was the active agent in the production of the symptoms. The hemianæsthesia which Mr. A. E. Berry observed in two of these men (Cases 2 and 3) has also been met with in other forms of toxic paralysis, such as lead and bisulphide of carbon poisoning, and it is probably a hysterical symptom induced by the deterioration of the general health, which results from the action of the poisons. At the Roburite Works, near Wigan, a workman died* soon after cleaning out a flue over the mixing room, which was said to contain di-nitro-benzene. The work was known to be dangerous, and the men engaged in it remained in the flue only three minutes at a time. This man was found in an insensible condition, and died on the following day.

(*f*)—THE PARALYSIS OF POISONING BY "ANILINE OIL."

Cases of acute poisoning in aniline works do not appear to be of uncommon occurrence, if the reports of the workmen are to be trusted, and it seems to be beyond doubt that the nitro-benzole or essence of mirbane used in the manufacture of the dye is the active agent in causing the symptoms. A very interesting case of this kind, under the care of Dr. Dreschfeld, has recently been reported by my friend Dr. Reynolds,† Medical Superintendent to the Infirmary, and although it is not within our province to discuss the symptoms of acute poisoning, I shall quote it at length, in order to fix attention upon the history the patient gave of the onset of his symptoms:—

"Francis O'B., aged 25 years, was brought into the accident room of the Manchester Royal Infirmary at 6-50 p.m. on Tuesday, March 19, 1889. He was quite unconscious and of an intensely blue colour, and the only history I could elicit from his friends was that he was employed in an aniline works, and that he had been sent home in the morning as sick, and with the instruction that he was not to be allowed to go to sleep. I diagnosed poisoning by nitro-benzole, admitted him, and at once took the following notes:—'The patient lies easily on his back, and is quite comatose, appearing, however, rather as if in a deep sleep. On applying a strong faradic current to the face he responds merely by a few grunts and grimaces. All the limbs are flaccid and helpless, the tendon reflexes being increased in some measure. The extraordinary feature of the case is the most intense blueness of the whole body, particularly the hands, feet, face, and mouth, including, of course, the tongue. This is more marked than any kind of cyanosis that I have previously seen. The eyes are closed, the pupils normal, reacting to light. The pulse is 100 and regular. The respirations are quiet (32), markedly Cheyne-Stokes in character. The breath has an odour exactly like aniline oil, but not at all like that of oil of almonds.'

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* See *The Lancet*, Vol. II., 1889, p. 81.

† Reynolds (Dr. Ernest S.). "Acute poisoning by nitro-benzole or essence of mirbane."—*The Medical Chronicle*, Vol. X., Manchester, 1889, p. 140.

"While I was examining the patient, Mr. Alcock, the house physician, touched his face, when he wakened up with surprising suddenness, and asked where he was. The Cheyne-Stokes breathing at once disappeared, and the respirations increased to 42 per minute. He complained of great drowsiness and of an intense, dull, aching pain in the head, and a feeling of heavy weight in the stomach. The blueness remained as before. The same night he did not sleep well on account of the headache, which, however, was somewhat better in the morning. On the 21st March he was much better, the cyanosis being but slight, though he still had some anorexia and cephalalgia. The urine was carefully examined. It was of a peculiar high colour, being turbid, and reddish-brown, specific gravity 1,032, contained no albumen, no blood, and no globulin. There was no reaction to Fehling's test, but a trace of bile was found ; it contained an excess of urea. It did not darken on standing. He gave me the following history :—He had been employed at an aniline works for seven weeks, making nitre cake and nitric acid, until Saturday, March 16th, when he went to 'picric' making. It was his duty to heat and 'whiz' a mixture which, he said, consisted of nitric acid, carbolic acid, vitriol, and mirbane, and the fumes from this mixture he constantly inhaled. He first felt sick, with pain in the stomach and headache, on Sunday, March 17th, and these symptoms were worse on the Monday, when he could hardly walk home. On Tuesday morning he was much worse, and reported himself as ill to the foreman, and the doctor came to see him, and gave him a quart of new milk and two eggs, and told him not to go to sleep. He went home in the course of the morning, and went to sleep in the afternoon. He was the only man engaged at this work. He further told me that one man had died from working with this mixture, and that no man could keep at the work for long together. The last man went home on Saturday morning, said to be dying, having been at the work for two or three months, being very pale for some time before he became blue.

"Francis O'B. was discharged from the hospital on March 21st, recovered."

For permission to publish the following case I am indebted to my colleague Dr. Steell :—

Case.—T. B., aged 22 years, entered the Royal Infirmary, under the care of Dr. Steell, on August 25th, 1890. The patient is a labourer in an aniline dyeworks, and is engaged in distilling aniline. While at work his hands are often smeared with naphtha and aniline, and in consequence the skin becomes brown and peels off in patches. He is also much exposed to the fumes from naphtha and from aniline oil, the latter of which he says is a mixture of nitro-benzole, nitric acid, and spirits of salts. In this mixture the nitro-benzole largely predominates, and the fumes from it are so strong that a workman is at times completely overcome by it, and has to be carried away in a partially unconscious condition, but is able to resume his work in from twenty minutes to half an hour. The workmen's lips become of a blue tint. This colour cannot be washed away, but gradually wears out in the course of a few days. Four weeks ago he began his work of distilling aniline on a Monday morning, and soon afterwards he felt sensations of numbness, and "pins and needles" in his toes and the tips of his fingers. These abnormal feelings gradually extended upwards, until at the end of the week they reached in the lower extremities as far as the hip joints, and in the upper as far as the shoulders. Three days later he was seized with a pain in the loins, and at the same time he felt very weak in his back.

The sensory disorders just described were, indeed, throughout accompanied by weakness. Almost at the same time in which the sensations of "pins and needles" began in the toes, he found that he had no use in them, and he experienced considerable difficulty in getting his clogs on, and felt tired on walking a few yards. He became, day by day, weaker, but kept at his work up to Friday night. On the following Monday, however, he could hardly stand, and could not raise his arms above the level of his shoulders, so that it was quite impossible for him to return to his work. At a very early period he began to suffer from painful "cramps" in the feet, which have continued to trouble him both day and night up to the present time. For the following fortnight the symptoms remained stationary, but at the end of this time improvement set in, and a week later, on his admission to the Infirmary, he could raise his arms above his head and stand without support, but could not walk. At first his lips were of a blue tint, but the colour gradually wore off, and disappeared at the end of about a week after he ceased from his work. The patient has not had sore throat, not been unduly exposed to cold and wet, never had syphilis, there is no blue line on the gums, and although he might take a few glasses of beer on a Saturday evening, he has never drank to excess, and hardly ever tasted beer or spirits during the week. He has never drank naphtha, but he smokes tobacco, and also chews about half-an-ounce per day. His father died some years ago, but he does not know the cause of his death. His mother, two brothers, and one sister are living and in good health. One sister died, but he does not know from what disease.

Present Condition.—The patient is a well-built young man of healthy appearance, except for the great pallor of his face. The conjunctivæ are pale and anæmic, but his lips are fairly rosy, and free from any blue tint. The only noteworthy symptom observed on the surface of the body is that the palms of the hands and the palmar aspect of the fingers are covered by a delicate epithelium, and the presence of old skin hanging in shreds to these surfaces shows that the old epidermis has peeled off in patches. The grasp of either hand is feeble, the left marking 25° and the right 20° on the index of the dynamometer, as compared with 80° by a healthy person. He cannot approximate the tips of the thumbs and little fingers without flexing the thumbs at the phalangeal joints. There is no wrist drop, but the power of extension of the fingers and at the wrist is feeble, and although he can perform the various movements at the elbow and shoulder joints, all of them are weak when tested by opposing actions. As the patient lies on his back in bed he cannot raise either heel from the plane of the bed when the legs are extended, and he has even some difficulty in drawing up each knee. The feet are somewhat dropped at the ankles, but there is no deformity in the position of the toes. He cannot raise himself into a sitting posture in bed without help, and when placed sitting on the edge of the bed with his feet planted on the ground he is unable to raise the ball of either foot from the ground, but can elevate the toes slightly. He cannot raise himself from the sitting to the erect posture without help, but once erect he can stand alone, but cannot walk without support. The abdominal and intercostal muscles, the diaphragm, and the muscles of the neck and face do not appear to be affected, but the tongue is tremulous on protrusion, and his voice appears occasionally to have a slight nasal quality, although he himself thinks it unaltered. The soft palate seems normal on inspection, but it does not contract readily on mechanical irritation, and the whole of the back of the throat is very tolerant to manipulation. The palate, however, has not quite lost its reflex contractility, and it also contracts during phonation. The bladder and rectum are unaffected. He complains of numbness and tingling sensations in his hands and feet, and a slight trace of anæsthesia is discovered on the distal segments of all the extremities when the sensibility of the surface is tested by pricking, double points, and hot and cold bodies, but the muscular sense appears to be normal. The calves of the legs are extremely sensitive to pinching, and it is found that the branches of the internal plantar nerve, particularly at the points where it bifurcates to form the digital nerves, are also very tender to pressure. His feet are hot and burning, and when they are allowed to hang over the bed in a dependent position they become of a dark purple colour.

The patellar tendon reactions are lost, the plantar reflex is sluggish, but the cremasteric, abdominal, and epigastric reflexes are active. The pupils are somewhat dilated, but they react to light and accommodation. The patient does not complain of diplopia, and there is no squint, nystagmus, or impaired movements of the eyeballs, and the optic discs are found to be normal on ophthalmoscopic examination.

The pulse is 68, regular, and compressible, but fairly full; the temperature is normal, the appetite is fairly good. A physical examination of the lungs, heart, and abdomen failed to elicit any sign deserving of being recorded. The urine is of an amber colour, specific gravity 1,016, acid, clear, and free from sugar or albumen. He was ordered a mixture of carbonate of soda and tincture of nux vomica, but on the following day the prescription was changed to a mixture of iodide of potassium.

August 27th.—The symptoms have continued unchanged since last report. The following galvanic reactions were obtained :—

		RIGHT.		LEFT.	
		K.S.C.	A.S.C.	K.S.C.	A.S.C.
Tibialis anticus	Cells Leclanché	30	35	20	35
Extensor longus digitorum	„ „	30	35	40	45
Peronei	„ „	30	40	40	45

The above muscles and the nerves supplying them react to a weak faradic current.

Sept. 24th.—The patient has improved considerably in general health since last report. He cannot, however, yet get up from a sitting to the erect posture without help, and although he can stand alone, is unable to walk a step without support.

Considerable doubt may reasonably be entertained as to how far we are justified in regarding this case as an after effect of poisoning by nitro-benzole. Passing over the difficulty which arises from the complexity of the fumes to which he was exposed, the great tenderness of the calves of the legs to pressure points to the case being one of alcoholic paralysis. But without giving undue credit to the statements of the patient himself, it is certainly most unusual for the paralysis of alcoholic excess to begin so abruptly as it did in this case without any premonitory symptoms, or to pursue such a rapid course except after such a severe bout of heavy drinking for many days or weeks, as there is no reason to suppose this young man to have been guilty of. He has not, moreover, suffered from the psychical symptoms of alcoholism. Another probable supposition is that the case is one of post-diphtheritic paralysis, and the presence of slight indications of paralysis of the soft palate lends considerable countenance to this view. On cross-examination the patient stated that, although liable to suffer from sore throat, he had his last attack eight months ago. He asserts positively that he has not had sore throat recently, or any other illness which prevented him from attending to his work. It will be hereafter shown that in epidemics of diphtheria paralysis may occur in persons who have not been known to have suffered from sore throat, just as in epidemics of scarlet fever, acute desquamative nephritis, with its attendant dropsy, is met with in persons who have not been known to have had a scarlet rash, sore throat, or fever, and it is quite possible, therefore, that this case is an example of post-diphtheritic paralysis in which the primary disease pursued a latent course. On the whole, however, I am inclined to reject this supposition and to believe that the symptoms have resulted from exposure to the fumes of nitro-benzole, an opinion which is much strengthened by the knowledge now ascertained of the action of this compound when used as an explosive agent in "roburite." The cases of nitro-benzole poisoning hitherto published describe only acute symptoms of the affection, and I shall consequently not refer to the literature of the subject here further than to direct the attention of the reader to the full bibliography given by Dr. Reynolds in his paper, from which I have already quoted.

IV. TOXIC MULTIPLE NEURITIS—(2) THE NEURITIS CAUSED BY ANIMAL POISONS.

(*a*) DIPHTHERITIC PARALYSIS.

THE record of the following case is chiefly drawn up from the notes supplied by the patient himself, Mr. John Dunlop, now one of the House Surgeons to the Royal Infirmary.

Case I.—John Dunlop, aged 24 years, had just been appointed, at the beginning of September, 1889, House Surgeon to the Manchester Royal Infirmary, and almost his first duty was to assist in performing tracheotomy on a child that had been brought in on the verge of suffocation from diphtheria.

On September 11th he woke up with a sore throat, and the glands of the neck were already swollen and tender to pressure. He got worse during the day, and on the following morning membranous patches were seen on both tonsils and on the soft palate. He was at once removed to Monsall Fever Hospital. The nurse who had assisted at the operation was seized in a similar way; she also was removed to Monsall, where she died a few days later from a malignant form of diphtheria.

September 16.—Since his admission to Monsall Hospital the whole of the soft palate, pharynx, and tonsils has become covered by an ash coloured membrane. The voice became gradually weaker, and the patient is now unable to speak except in a whisper. The pulse is 120, regular and of fair strength, and the temperature has varied from 102°F. to 103° F.

September 20.—The soft palate and pharynx are now free from membrane, but there is still an ash coloured slough on each tonsil. The big toes are slightly flexed into the soles, and fluids regurgitate through the nose. The patellar tendon reactions are sluggish, but still present. The patient complains of seeing objects double, and a slight squint is observed.

September 24.—The patient is now convalescent, and the throat, although somewhat red, is free from membrane. The temperature is normal and the pulse has fallen to 90 beats per minute, but the patient is still very feeble. He is unable to speak above a whisper, and the skin over the abdomen is very sensitive to pinching.

October 7.—The patient is still improving and his appetite is good. There is also some sign of improvement in his voice, but it is at times deep and hoarse, and at other times high and squeaky, and is under imperfect control.

October 13.—To-day he has felt for the first time a numbness and tingling of his finger tips, these symptoms being most pronounced when he washes his hands.

October 20.—Fluids have regurgitated through the nose this morning, and it is found that the soft palate is insensible to touch, and does not contract on mechanical irritation or during phonation. The voice is nasal. The tingling and numb sensations have now extended to the toes and have become more marked in the fingers.

October 26. The voice is still nasal and fluids regurgitate through the nose on swallowing. The abnormal sensations in the hands and feet have not yet abated, but as the patient is otherwise in excellent health, though weak, he is allowed to go to his home at the seaside.

November 1.—Fluids still regurgitate through the nose and the voice is nasal. On swallowing, liquids are liable to pass into the larynx and give rise to paroxysms of cough. The numb sensations experienced in the hands and feet have now extended up the forearms and legs.

November 7.—Visited Dr. Ross. The voice is still nasal, and fluids regurgitate through the nose. The soft palate is insensible to touch, and does not contract on phonation or on mechanical irritation. The knee-jerks are present. A slight squint is observable, but the patient does not complain of diplopia, and he can read a little, but soon tires. The vocal cords are found on laryngoscopic examination to act during inspiration and phonation. The numb sensations already described extend up as far as the elbows and knees; but there are no manifest sensory disorders to subjective examination. When each foot is unsupported, it is seen to drop a little at the ankles, and the big toes are hyper-extended at the proximal and extended at the distal joints; but none of the movements at the ankle or of the toes are manifestly paralysed. The gait is feeble and uncertain, and the toes show probably a slight drop when the foot is advanced in walking, but not sufficient to give to the gait a high-stepping character.

November 15.—The voice has now lost its nasal quality, and fluids can be swallowed without inducing a paroxysm of cough or regurgitating through the nose. The numb sensations in the extremities have become more marked. The knee-jerks are lost.

November 26.—Saw Dr. Ross to-day. The knee-jerks are lost. When seated on a chair with his feet flat on the ground, he is scarcely able to raise the ball of either foot from the ground. He cannot stand on tiptoes. The gait has a distinctly high-stepping character.

November 30.—The fingers are very numb, and tactile sensibility in them is so much impaired that in adjusting his clothes he is hardly able to distinguish without looking whether or not he gets a hold of the buttons. The fingers are also feeble, and he consequently experiences great difficulty in buttoning his clothes. He cannot ascend a stair without assisting himself by holding the banisters.

December 3.—Saw Dr. Ross again. The knee-jerks are lost. Each foot, when unsupported, is manifestly dropped at the ankle, and the big toes are hyper-extended at the proximal, and flexed at the distal, joints. When the patient sits, and his feet are planted on the ground, he can raise the inner edge of each foot slightly from the ground by the action of the tibialis anticus, but he cannot raise the outer edge in the slightest. When the patient stands, his feet are kept wide apart, and he sways from side to side on closing his eyes. The gait is staggering and uncertain, but presents a peculiar modification of the high-stepping action. The foot about to be advanced is quickly raised, in such a way that its inner border, and with it the ball of the toe, is jerked upward, while the small toes linger for a moment on the ground. During the advance of the foot the inner edge of the foot and big toe are held on a higher level than the outer edge and little toe, and when the foot is brought to the ground, the ball of the big toe comes down with a thump, as in locomotor ataxia. When the patient walks with uncovered feet, the tendon of the tibialis anticus is seen to start into prominence at each advancing step, and it is manifest that the modification of gait here observed depends upon the fact that this muscle has partially recovered, while the peroneal muscles are still paralysed. A slight squint is occasionally observed, but the patient does not complain of diplopia. He was now ordered to maintain the recumbent posture, either in bed or on a couch, and to have massage.

January 6, 1890.—Since the date of last report the patient has steadily improved in strength. The knee-jerks can now be elicited, although they are feeble and inactive.

January 14.—Saw Dr. Ross again. The patient can now produce dorsal flexion of each foot, and is able to stand for a short time on the tips of his toes. The big toes are in the normal position of extension at both joints. The gait is normal, or nearly so, and the knee-jerks are fairly active. The pulse, which beats 75 in the minute when the patient is quiet, becomes accelerated to 95 beats when he walks two or three times across the floor. The patient now regards himself as having completely recovered, although he is soon fatigued on exertion, and no further notes of his case were taken.

The following case is described in considerable detail, because the paralysis has pursued a fairly typical course, and, because the patient being under observation at the present moment, an opportunity has been afforded for observing the symptoms with accuracy. The notes were taken by my clinical clerk, Mr. J. H. Taylor.

Case 2.—E. S., aged 19 years, was admitted as an in-patient to the Manchester Royal Infirmary on September 4, 1890. The patient is by occupation a boatman on one of the canals in the neighbourhood of Manchester, and as he has lived on a canal boat for many years he has necessarily been much exposed to all kinds of weather, often on defective food, and imperfectly clad. He is almost an abstainer from alcohol, but smokes between four and five ounces of twist tobacco in a week. He has never suffered from any venereal affection, or from rheumatism. He has been told that he had a severe attack of croup in childhood, but he remembers nothing of it himself. He has also suffered from occasional headaches and diarrhœa, sometimes felt a pricking pain over the region of the heart when walking fast, and has at times had a slight cough, but without being accompanied by expectoration. He does not, however, remember to have been laid up with any serious disease until the commencement of his present illness. His mother died many years ago from cholera, and his father died from inflammation of the lungs, while an aunt is said to have died from consumption. He has had no brothers or sisters.

The patient is a well built young man, of average size, and fairly healthy appearance. He states that he was enjoying excellent health until nine weeks ago when, after exposure to wet and cold, he began to suffer from a sore throat, and stiffness of the neck. The attack was ushered in by shivering, which gave place in a few hours to profuse perspiration. His throat was very sore, especially when he attempted to swallow, and a swelling appeared on the left side of the neck behind the jaw. He also felt very weak and ill, and remained in bed for three days, during which he treated himself with "barley-water gargles," and warm flannels round the neck. He was troubled with a cough and expecporated a considerable quantity of a thin phlegm, stained of a pinkish colour, but he did not notice any shreds of membrane in it. He is certain that the phlegm came from the back of the throat. After lying three days in bed he got up, but felt very weak, and a few days later he consulted a doctor, who told him he had had a "quinsey," and ordered him some medicine, a gargle, and the application of poultices round his neck. About the middle of July, or a fortnight after the commencement of his illness, he became an out patient at the Infirmary, and was seen and prescribed for by Dr. Harris. All pain had now subsided for some days, but he began to experience a difficulty in swallowing—in his own words, "the food would not go down however much I tried"—and he was consequently compelled to restrict himself to fluid nourishment. But he soon found that he had a difficulty in swallowing fluids also, and whilst drinking a cup of tea most of it spirted out of his nose, and the medicine was ejected in the same way. The only way in which he could swallow a little was by throwing his head well back and allowing the fluid to trickle down. The difficulty in swallowing lasted only three days before improvement set in, and at the end of a week he could again drink with ease, but he had some difficulty in swallowing solids for a day or two longer. This brings us down to July 24th, and on August 1st fluids began again to be ejected through the nose, and he also found that both solid food and liquids "went down the wrong way," and caused violent fits of coughing. These symptoms have continued up to the present, but the difficulty in swallowing varies in degree from day to day; sometimes he can swallow with tolerable facility, but at other times fluids return by the nose, and solids seem to stick in his throat. At this date he noticed, for the first time, that his speech was altered in quality, and this symptom has persisted, without any variation in degree, up to the present.

About a fortnight ago, or seven weeks after the commencement of the sore throat, his vision became indistinct. A mist seemed to be in front of him, and he found he could no longer read because "the letters ran into one another." By making a great effort he could see the printed type of a newspaper for a few seconds, but the mist came again between his eyes and the page, and he was obliged to desist from all attempts at reading. A week later he was surprised to find that the one horse, which he was at the time driving, had suddenly appeared as two. His eyes began at the same time to ache, and he had a feeling of dizziness and uncertainty in walking. He also noticed that the tips of his fingers were of a bluish colour and wrinkled, just as if they had been for a long time in water. Two days later he had a feeling of numbness in his fingers, and at the same time he suffered from attacks of cramps in the calves of the legs, which were soon followed by numbness of the feet, accompanied by a sensation as if the toes were being squeezed. The numb sensation in the hands gradually crept up as far as the elbows, and at the same time the grasp of each hand became very feeble. At the end of another week the numbness began to recede, and, on admission, it was again restricted to the hands.

The numbness of the feet gradually ascended the legs and thighs, and soon reached as far as the hip-joints, while his legs became at the same time so weak that he could hardly stand. He also states that his limbs have wasted considerably since his illness began. On the Monday (Sept. 1st) before admission, he was walking along the towing-path of the canal, holding on to a horse, when suddenly he felt a pain over his heart, and his legs gave way under him, but he believes that he "did not faint on falling." The pain ceased in a few minutes, but he felt so weak that he had to be carried home. He now found that he was becoming day by day more helpless, and entered the Infirmary as an in-patient.

Present Condition.—The patient is a well-built young man, and fairly nourished, although his face is somewhat sallow. His expression and the surface of his body generally do not present any peculiarities deserving of note. As the patient lies in bed the first symptom to attract attention is that his voice has a nasal quality, and on enquiry it is found that, on swallowing, fluids are ejected through the nose. He cannot puff out his cheeks or blow out a candle, and when he endeavours to perform these actions air escapes through his nose. Solids can now be swallowed with facility. The whole of the back of the throat is very tolerant to manipulation, and the soft palate does not contract on mechanical irritation. It does, however, contract slightly on phonation. On laryngoscopic examination, the vocal cords do not present the pearly lustre of health, but are of a dull reddish colour. They move freely, however, during deep inspiration and vocalisation. The patient is extremely tolerant of the laryngoscopic mirror, and the bifurcation of the trachea can be readily seen, but on touching the posterior surface of the epiglottis with a bent piece of wire, the glottis immediately closes and the epiglottis is depressed, while a cough and tendency to vomiting are induced.

There is a slight want of convergence in the eyes when the patient's gaze is fixed upon an object, but he is not much troubled with double images. When, however, a red glass is placed in front of the right eye, a lighted taper in front is seen double, the false image being to the right, and when the glass is placed in front of the left eye, the false image is seen to the left, so that it is probable there is some relative feebleness of both the external recti muscles. There is also slight nystagmus on lateral movements of the eyes to either side. The patient can read for a short time medium-sized print by holding it at a distance of twenty inches from his eyes, but by the use of No. 20 convex lenses he can read easily at a distance of ten inches. The pupils are somewhat dilated, but are equal, and react to both light and accommodation. On ophthalmoscopic examination, the optic discs are found to be normal. The grasp of either hand is very feeble; by the right he moves the index of the dynamometer 25°, and by the left only 12°, as compared with from 90° to 100° by healthy persons. When the patient makes an effort to grasp strongly the hand becomes flexed to nearly a right angle at the wrist, and it is also pronated. He cannot appose the tips of the thumbs and little fingers without flexing the thumbs at the phalangeal joints. All the movements at the elbows and shoulder joints are feeble, but no individual muscle or group of muscles appears to be affected in special degree.

As he lies on his back in bed, the patient can perform the movements of the lower extremities, including those of the toes, but all of them are lacking in force. He can, for example, raise the heel of each foot from the plane of the bed, but the slightest resistance suffices to arrest the movement. The foot is somewhat dropped at the ankle, and the big toes are slightly flexed at both joints; but the patient can produce dorsal flexion of the feet and extension of the big toes by voluntary effort. When he sits on a chair with the feet flat on the ground, he can raise the ball of the toes from the ground by voluntary effort, but during this action the foot is seen to be slightly inverted, owing to the predominance of the action of the tibialis anticus muscle. All the movements at the ankles, knees, and hip-joints, as tested by opposing actions, are feeble, and of these extension of the legs upon the thighs is most affected. The patient is able to extend each leg upon the thigh against the action of gravity, but the slightest pressure on the anterior surface of the leg when the thigh is fixed, suffices to overcome the resistance.

The patient can raise himself in bed with considerable difficulty by turning to one side and pressing against the bed, first by his elbow and then by his hands. When, however, he is seated on the edge of the bed, he experiences great difficulty in attaining the erect posture, and he can only do so by clutching at some object with his hands, or by climbing up his thighs, as in pseudo-hypertrophic paralysis. When once erect, he can stand alone, but has to carefully balance himself. His feet are then wide apart, and the body is thrown back, so that a plumb-line let fall from the most prominent of the dorsal vertebræ, clears the sacrum by half-an-inch. When he closes his eyes, his body sways from side to side, and he would fall unless supported. He cannot walk a step unless he is supported on either side by an assistant. When so supported, he throws his buttocks back and his body forwards, so that the weight of the trunk is borne by the assistants, and he now slides each advancing foot forward, the heel being only raised about an inch, and the toes scraping the ground. At each step the knee on the side of the foot planted on the ground manifests a tendency to jerk forwards, so that, unless well supported, the patient would fall to the ground by his legs doubling up under him.

The abdominal muscles act strongly when the patient is asked to raise himself in bed, but when he is asked to protrude the abdomen, as in straining, the action of the diaphragm is found to be feeble, although the muscle is not paralysed. The difference in the circumference of the chest, on a line with the nipples, between maximum inspiration and maximum expiration is 1¾ inches. The patient has to strain for a long time before he begins to urinate, and the act costs him a great effort, while the stream is lacking in force, but there is no dribbling. The patient also suffers from great constipation.

The patellar-tendon reactions are absent, but the plantar, gluteal, cremasteric, abdominal and epigastric reflexes are active. The reflex of the palate has already been mentioned as absent.

The patient complains of a feeling of numbness of the upper extremities which extends as far as the elbows, and of the lower as far as the hip joints. On objective examination the fingers, on their dorsal and palmar surfaces, and the palms of the hands are found to be completely insensible to the prick of a pin, but the backs of the hands and the forearms are acutely sensitive to pricking and pinching. On the outer surfaces of the forearms two points are felt as one until they are removed four inches apart. Hot and cold bodies are readily distinguished with closed eyes on the forearms and backs of the hands, but not on the palms of the hands and fingers. The patient, when his eyes are closed, is unable to tell the position in which his fingers are placed, but he can tell accurately the position occupied by the hands, forearms, and arms. In the lower extremities the feet and toes, as far as the ankles, are completely insensible to the prick of a pin, while the patient is not able to tell, with closed eyes, the difference between the touch of the head and the point of a pin from the ankle up to the middle of the calf. On the outer surface of the legs two points are felt as one until they are removed five inches from one another. The patient cannot tell, his eyes being closed, the difference between the touch of a hot and a cold body on the feet; he can distinguish correctly above the ankles, but from the ankles to the middle of the calves there is a delay of several seconds between the moment of contact and the patient's appreciation of the difference. He can tell with tolerable accuracy, his eyes being closed, the position in which the lower extremities may be placed, with the exception of the feet and toes, of the position of which he has little or no appreciation. The cutaneous electrical sensibility is diminished in the palms of the hands and fingers, and in the feet and toes.

The nerves and muscles most affected react to a faradic current of moderate intensity, the faradic irritability, as compared with that of healthy persons, being probably somewhat impaired but nowhere lost. The soft palate is very tolerant to the faradic current, and a strong current is required in order to obtain a minimum contraction.

The following galvanic reactions were obtained :—

		Right.			Left.		
		K.S.C.		A.S.C.	K.S.C.		A.S.C
Arms—Extensor communis digitorum	Cells Leclanché	25		25	25	>	25
Extensors of the thumb	„ „	25	>	25	25	>	25
Legs—Quadriceps extensor	„ „	20		20	20		20
Tibialis anticus	„ „	25		25	20		25
Extensor communis digitorum	„	30		30	25	<	25

The tongue manifests a considerable degree of diminution of ordinary sensation, the prick of a pin being felt as a touch and not as painful. The patient did not perceive the taste of salt rubbed on the tongue until he swallowed, and he quite failed to appreciate the bitter taste of quinine. The sense of hearing is also somewhat impaired, the tick of a watch being only heard at a distance of six inches. The sense of smell is normal, and the state of vision has already been described. The tongue is clean, and the patient's appetite is good, and, with the exception of constipation, his digestion may be said to be normally performed.

The chest is found to be free from any sign of disease on physical examination. The heart is normal in size and position as determined by palpation and percussion, and the sounds are clear and free from murmur. The pulse is regular, very compressible, and beats 70 when the patient is tranquil and in the recumbent posture, but it rises to 102 beats in the minute on the slightest exertion.

The patient was ordered five minims of liq. strychnia three times a day, rest in the recumbent posture, and nourishing diet.

September 7.—The bowels not having been moved, the patient was ordered on the morning of the 6th a black draught, and as no relief had been obtained, he had a dose of castor oil this morning. A few hours later he suffered from griping pains in the abdomen, and was smartly purged. On getting up to the night chair he suddenly fell to the ground in a faint, and was unconscious for a short time. During the attack the pulse was feeble and frequent, and at the usual daily visit it beat 90 in the minute, and was feeble and compressible, but regular.

September 8.—The pulse is stronger to-day, and now beats 84 in the minute. He still complains of griping pains in the abdomen, but has not been again purged. His symptoms have continued unchanged in other respects.

September 12.—His nose bled a little to-day without any obvious cause, but there is no other noteworthy change in his condition.

September 20.—The patient has improved greatly in general health since last report. His voice has lost its nasal quality, and, on swallowing, fluids are not returned through the nose. His bowels are less obstinately constipated than they were, and he can void his urine without unusual straining or effort. His grasp is, however, still feeble, and he cannot walk without support. The patellar tendon reactions are absent. He now complains of having occasional attacks of cramps of the ulnar fingers of each hand. The toes also are frequently drawn by cramps, and the patient states that the crampy sensation is limited to the soles of the feet, and does not extend to the calves of the legs. It is thought possible that the strychnine he is taking for medicine may contribute to induce these cramps, but as the symptom is in no way urgent, no change is made in the treatment.

September 22.—He can now stand, but is unable to walk without support. The cramps are less severe than they were, and consequently no change is ordered in his medicine.

September 25.—The patient can now raise himself into a sitting posture in bed without the assistance of his hands, and he can also stand and walk feebly without support. He is in excellent health and spirits, and is manifestly advancing rapidly to complete recovery.

In the following case the patient—a distinguished professional brother—supplied the notes, but they are supplemented by my own observations :—

Case 3.—Mr. T. S. S., aged 49 years, was first seen by me on January 9, 1888. He has always enjoyed excellent health, but in 1886 he was much alarmed one morning on finding that he could not close his right eye. A few days later I saw him, and it was evident that he was suffering from a slight degree of paralysis of the right facial nerve. It was not accompanied by loss of taste of the right half of the tongue, and it was consequently manifest that the nerve was affected either at the lower end of the Fallopian canal or after its emergence from this canal. The symptoms came on after the right side of his face had been exposed to a draught of cold air when he had been heated, and complete recovery took place in two or three months without leaving any deformity behind. In November of 1887, he was attending several cases of diphtheria, and he subsequently remembered that about the end of the month he himself had a slight sore throat, but it was not attended by the formation of membrane or by ulceration, and he attached no importance to it at the time.

Some time in December he found that his eyesight failed somewhat suddenly, and he was obliged to use convex glasses when reading or writing, but this he attributed to rapidly advancing presbyopia, although his eyesight had previously been very good. There was no double vision. During the last two weeks of December he felt unequal to his work, and in the first week of January, 1888, he experienced, for the first time, distinct difficulty in walking. He also felt great mental depression, and was troubled by shooting pains in the palms of the hands and thenar eminences, which extended to the tips of the middle fingers. These pains were more marked in the left than in the right hand. He had at no time any difficulty in swallowing, and did not himself recognise any change of voice, but a friend afterwards told him that whilst conversing with him one day he observed that his voice had a peculiar nasal twang.

January 7, 1888.—To-day he felt for the first time tingling sensations, like "pins and needles," in the toes and soles of the feet, and in the tips of the fingers. He also experienced a feeling of great fatigue as the day advanced, but in the evening he walked half a mile to see a patient, and thought himself rather better for the exertion. On returning home, however, he was alarmed by finding that both knee-jerks were absent.

January 8.—On getting up this morning he found he could hardly stand, and this weakness rapidly increased during the day, so that in the evening he could not walk across the floor without being supported on each side.

January 9.—The tingling sensations in the extremities increased in severity, and now extended up the forearms and legs. The tactile sense is much diminished in the tips of the fingers, and paper feels like felt. On closing the hand the fingers feel enormously swollen, although they are in no way enlarged when examined. The prick of a pin can be readily felt in the hands and forearms. On placing the feet on the floor there is a sensation as if cotton wool were interposed. The grasp of either hand is very feeble, that of the right being the weaker of the two. On attempting to grasp strongly the forearm becomes pronated, and the hand strongly flexed at the wrist, showing a predominance of the paralysis of the extensors and supinators over that of the flexors and pronators. All the movements of the forearm and arm are weak, and accompanied by so much tremor and inco-ordination that the patient is unable to feed himself. The feet are somewhat dropped at the ankles, and the big toes are hyper-extended at the metatarso-phalangeal joints. All the movements of the lower extremities can be feebly executed as he lies in bed, but he is unable to exercise any degree of force against opposing actions. He is also unable to raise himself in bed without help. The movements of respiration are not appreciably affected. The palpebral aperture of the left eye is slightly larger than that of the right; but it was thought possible that the difference in size of the apertures was the result, not of a widening of the left from weakness of the orbicular muscle, but of a slight contraction of the right aperture, which was left as a sequel of the old facial paralysis. The temperature is normal; the pulse beats 100 in the minute, and is very compressible. The urine contains phosphates, but is free both from sugar and albumen.

January 10.—To-day the patient has experienced considerable difficulty in voiding his bladder, and also in straining at stool. When he is asked to raise himself in bed, the hand of the observer placed over the abdomen feels the abdominal muscles contracting well; but when he is asked to strain, as if at stool, the abdomen is momentarily protruded and then relaxes, to be followed by another protrusion and another relaxation. The feeling given to the hand of the observer placed on the abdomen is that of a clonic spasm of slow rhythm of the diaphragm. There is a slight falling in of the abdomen during inspiration. The difference in the circumference of the chest, on a level with the nipples, between maximum inspiration and maximum expiration, is only one inch. There is no dyspnœa, but the patient complains of a feeling of oppression and of constriction round the chest. All the extremities are now almost completely powerless, and they lie like inert masses where they are placed on the bed. The left lower eyelid is on a slightly lower level than the right; it is somewhat everted, so that tears collect between the margin of the lid and the eyeball. The left upper eyelid is drooped, so that it almost entirely covers the pupil. The right eyelid appears to act in a normal manner. There is also a slight internal squint of the left eye. The pupils are somewhat dilated, but are equal, and contract to light and accommodation. The patient is troubled with attacks of profuse perspiration, which come on several times during the day, and the secretion has a sour odour, like that of rheumatism. He is also much troubled with considerable flatulent distension of the bowels. He was very restless during last night, and was obliged to have a chloral draught in order to secure a little sleep.

January 14.—The patient has continued much in the same condition since last report. He has had very restless nights, and has complained much of a sense of constriction and oppression about the chest. To-day, however, it is found that the diaphragm is maintained in a state of uniform contraction when he is asked to strain, and the difference between the circumference of the chest on maximum inspiration and maximum expiration is 1½ inches. He still suffers from marked constipation, and the bowels have to be relieved by enemata. Micturition is likewise hesitating and ineffectual. There is slight recovery of motor power in the left arm and left leg, and to a less extent in the right leg also, but the right arm continues as helpless as ever, and he experiences in it a sensation of great enlargement and tension, although it appears quite normal in external appearance. The patellar tendon reactions are absent. When the patient's eyes are directed horizontally forwards or slightly downwards, nothing abnormal is observed in the positions of the eyeballs or eyelids, with the exception of a slight internal squint of the left eye, but when he tries to look upwards, the upper eyelids become strongly retracted, while the eyeballs rotate little if at all beyond the horizontal level. The consequence is that a large portion of the white sclerotic is exposed above the corneæ, and this gives to the eyes a wild and staring expression. This deformity appears to be caused by the fact that the branches to the levator palpebræ have recovered at least relatively, while the superior recti muscles are paralysed, so that the effort to direct the gaze upwards is effective with the former and inoperative with the latter set of muscles. The left lower eyelid is still on a somewhat lower level than the right, and tears accumulate between it and the eyeball, but beyond some loss of general expression of the face, there is no further evidence of implication of the facial nerves.

January 21.—All the extremities have regained a considerable degree of motor power, and even the movements of the right upper extremity, which lags behind the other limbs in its recovery, can now be made by voluntary effort at all the joints, except at the proximal and distal joints of the thumb, the power of extension at which is still lost.

The sensation of tension and swelling in the right arm has gradually disappeared, and in place of it there is a feeling as if the limb were loaded with heavy weights. During the last week great pain was felt for the first time along the inner and anterior surfaces of the right arm and forearm, and the skin over this area has been exquisitely sensitive to the slightest touch. The pain is aggravated to a pitch of high intensity by extension of the forearm, but it had its seat on the surface of the limb, and not in the joint. A cutaneous branch of the internal cutaneous nerve can be felt like a piece of whipcord passing from a little below the elbow down the front of the forearm to near the wrist; it is excessively tender to touch, but the skin over it is not discoloured. There is also a considerable degree of hyperæsthesia of the palmar surface of the index finger of each hand, but the numb sensation previously felt in the fingers has in great part disappeared from the middle, ring, and little fingers of the left hand, but persists in the corresponding fingers of the right hand. He it still subject to periodical attacks of profuse perspiration, each attack being preceded by a feeling of heat and fever, but without actual elevation of temperature, and followed by great mental depression. To have treatment by massage.

January 31.—Continued steadily to improve in muscular power since last report. Can now move the limbs freely in bed, with the exception of the right arm, which is as yet very feeble in all its movements. On attempting to rise in bed the dorsal muscles feel to the patient as if they were in a state of great rigidity.

February 7.—He is now able to rise to a sitting posture without extraneous help, and can move the limbs freely in bed, with the exception of the right arm, which still continues heavy as well as tender to touch, uneasy when at rest, and exquisitely painful on the slightest movement. The urine is expelled with force and without conscious effort, and the bowels are less obstinate than formerly.

February 20.—He has continued to improve day by day, and is now able to sit in a chair from three to four hours during the day, and to walk across the room with a little assistance. The soles of the feet have still a "padded" feeling when planted on the ground. The pain in the right forearm is now limited to the flexor surface of the forearm and the palmar surface of the hand, but the back of the forearm is still tender to pressure. These pains are very troublesome at night, and cause much loss of sleep. The palmar surface of the index finger of the left hand continues to be in a state of hyperæsthesia. The patellar tendon reactions are absent. The patient now continued to progress uninterruptedly towards recovery, and a week later he went to the seaside for change of air.

The patient was seen by me again about the beginning of April, and with certain minor exceptions to be immediately mentioned, he had then made a complete recovery. The sensory disorders had disappeared, with the exception of a slight feeling of numbness of the palm and finger tips of the right hand, which became well marked on exposure of the hand to cold. The skin of the fingers of this hand was somewhat glossy as compared with that of the fingers of the left hand, and the patient was unable to extend the thumb fully at either the metacarpo-phalangeal or phalangeal joint. He was also unable to raise the right arm beyond the horizontal level, and on attempting to do so the inner border of the scapula was drawn towards the vertebral column, and the inferior angle was removed from the chest, the deformity resulting from paralysis of the serratus magnus. The patellar tendon reactions had now reappeared, but the reaction was still feeble.

He was next seen by me at Midsummer; he could now raise the right arm to a vertical position, and all deformity about the shoulder blade during the action had disappeared. He had also recovered from all abnormal sensations in the right hand, but the right thumb was held in a position of slight flexion at both the metacarpo-phalangeal and phalangeal joints, and the patient was not able to extend it fully at either joint. It will thus be seen that the only remnant of his former symptoms now to be found was a slight degree of feebleness of the proper extensors of the thumb of the right hand. The next time he came under my observation he had fully recovered from this slight disability, and he has since remained in perfect health.

When acting as visiting physician to Monsall Fever Hospital in the months of July, August, and September, last year, Dr. Bowman, then assistant resident medical officer to the institution, kindly took notes for me of all the cases of diphtheric paralysis which occurred in the hospital during this time. During these months 90 cases of diphtheria were admitted into the wards, and in 20 of these unmistakeable, if in some cases slight, signs of paralysis were noted. It would serve no useful purpose to publish the notes of all these cases, but I select a few of them which may be taken as fair samples of the rest.

Case 4.—E. H. B., aged 6 years, is admitted on June 8, 1889. The patient is feverish and complains of sore throat, and on inspection the fauces are seen to be covered by white membranous patches. The patient passed through a moderately severe attack of diphtheria without any noteworthy symptom having developed except great general debility, and it was not until the beginning of July that the first sign of paralysis showed itself. It was then found that the patellar tendon reactions had disappeared and that the big toes were flexed into the soles at the terminal phalanges, and slightly hyper-extended at the proximal ones. The plantar reflexes were present, but sluggish. No sensory disorders were complained of and none were discovered on objective examination.

July 15.—The voice is distinctly nasal, and on inspection the soft palate is seen to be somewhat pendulous, but it is drawn slightly backwards on phonation, and the uvula is in a symmetrical position. There is, however, little or no contraction of the palate on mechanical irritation. The ocular movements are normal, and the pupils, somewhat dilated, react to both light and accommodation.

July 29.—For the last few days a slight degree of internal strabismus has been noted, but it is intermittent. The voice is still nasal in quality, but the soft palate moves fairly well on phonation, although its reflex irritability is still lowered. The face has lost much of its expression, and when the patient closes her eyes a small rim of the sclerotic and cornea is seen between the lids on each side. She can only offer a very feeble resistance to the raising of the upper eyelid by the finger of the observer. The naso-labial folds are obliterated, and they do not become well marked when the patient smiles or endeavours to show her teeth. The grasp is weak, and all the movements of the upper extremities are feeble, but no muscular group appears to be affected in special degree. The patient can stand and walk across the floor unsupported, but the gait is tottering and feeble, and she is in danger of falling at every step. A few days later the patient was discharged from the Hospital, and no further opportunity was afforded for noting the progress of the case.

Case 5.—A. F., aged nine years, was admitted on July 15, suffering from diphtheritic sore throat. The sore throat pursued a favourable course, and the patient was convalescent, although still feeble, at the end of ten days.

July 30.—The knee-jerks are absent, and the great toes are flexed at both joints.

August 5.—Fluid returned through the nose this morning for the first time, the knee-jerks are still absent, and the cutaneous reflexes are diminished. The voice is nasal.

August 20.—The patient is now unable to read in ordinary type, but can make out a few words in large type. Regurgitation of fluids through the nose is only occasionally present. The knee-jerks are still absent, and the patellar tendons, when struck with the ulnar border of the hand, are felt to be toneless. The patient can now stand unsupported, and she walks a few steps across the floor, but her gait is feeble and uncertain. The following electrical reactions were obtained. A much stronger faradic current is required to produce a minimum contraction in the anterior tibial muscles than in the corresponding muscles of a healthy child. Galvanic reactions:—Anterior tibial muscles. Cells Leclanché—KSc. = 25, KOc., none, 50, A.Sc. = 25, AOc. = 30. Similar reactions were obtained in the extensors of the forearms.

August 30.—The patient can now read in ordinary sized type.

September 3rd.—The knee-jerks have reappeared, but only a very feeble contraction of the quadriceps femoris is obtained on tapping the tendon. The electrical reactions are practically the same as those obtained on August 20, the anodal being equal to the cathodal closing contraction in the anterior tibial muscles and in the extensors of the forearms. The patient was now discharged.

Case 6.—C. A., aged 24, was admitted as an in-patient to the Monsall Fever Hospital on August 22, 1889. The patient is feverish, complains of sore throat, and the fauces and uvula are covered by patches of white membrane.

September 12.—The patient has been convalescent for about a week, but to-day she complains of numbness and tingling in her hands, the two ulnar fingers, and the ulnar borders of the hands and forearms being the parts chiefly affected. The grasp of either hand is somewhat feeble, but the special movements of the fingers are not appreciably affected.

September 19.—The grasp of either hand is considerably feebler to-day than at the date of the last report. The knee-jerks, although still present, are very sluggish this morning, and the big toes are hyper-extended at the metatarso-phalangeal joints, while the feet are slightly dropped at the ankles.

September 21.—The big toes are flexed towards the sole at both joints. The following reactions to the galvanic current were obtained:—Leg—Anterior tibial muscles, cells Leclanché: Right—KSc. = 25, ASc. = 25, KOc., none, 50, AOc. = 30. Left: KSc. = 25, ASc. = 25, KOc., none, 50, AOc. = 30.

September 23.—Fluids regurgitated through the nose this morning for the first time, and the voice has a nasal quality. The reflex irritability of the soft palate is diminished.

September 28.—For the last two days fluids have ceased to be ejected through the nose, but the patient has been obliged to give up her needlework owing to failure of sight. The pupils are somewhat dilated, but react to light and accommodation.

October 4.—The patellar tendon reactions are now active, and vision is normal, the impairment of it having only lasted three days.

October 11.—No further symptoms have appeared since last report, and the patient was now discharged.

Case 7.—A. B., aged 10 years, was admitted to Monsall Hospital on December 4, 1889. Her throat was covered by an ash-coloured membrane, but at the end of ten days the throat cleared and the patient was fairly convalescent, although too weak to leave her bed.

December 17.—The patient, on sitting up in bed to take some milk, fell back and died in about ten minutes.

Case 8.—F. P., aged 21 years, had a sore throat whilst nursing in the diphtheria wards. A week after convalescence was established she experienced great weakness in trying to walk, or even in sitting up. It was now noticed that her pulse, which beat 80 in the minute when the patient was lying down, suddenly got up to 130 in the minute on sitting up in bed. This symptom only lasted three days, and no other sign of paralysis was noted.

Case 9.—A. F., aged three years, was admitted to the Hospital on November 7, 1888. The breathing was so much embarrassed that tracheotomy had to be performed on the following day.

November 26.—Since the operation the progress of the case has been fairly favourable, but to-day it is noticed that a portion of the fluid swallowed appears occasionally at the wound in the trachea and causes paroxysms of suffocative cough. Whilst feeding the child it was found necessary to place a plug of cotton wool in the wound in such a way as to intercept the passage of fluid into the trachea, and so long as this plug was held in position no cough was excited, but immediately on its removal the smallest drop of fluid passing into the trachea provoked a violent paroxysm of cough. This condition lasted for a week, when the child died from empyema. The absence of cough when fluids were prevented from passing into the trachea showed that the interior surface of the larynx and of a few of the upper rings of the trachea were completely anæsthetic.

In addition to furnishing me with notes of cases, Dr. Bowman has drawn certain conclusions from his facts, which possess at least that freshness which always characterises inductions from personal observations, and are in the results attained of sufficient importance to warrant reproduction here.

(1) *The relative frequency of paralysis after diphtheria.*—Most of the

cases of diphtheria are discharged from Monsall Hospital as soon as they are sufficiently convalescent to walk and to take their food well; and, consequently, if a patient recovers quickly from the primary affection no opportunity is afforded of observing whether or not paralysis is subsequently developed. Taking those cases which have from any cause been kept in the hospital for more than three weeks, and those which I have been able to follow after their discharge, I have only met with rare instances in adults that had not manifested some form of paralysis, however transient. A few cases which were under observation for a month and upwards were, however, entirely free from any trace of paralysis.

Children do not appear to be so frequently attacked by paralysis as adults. It must, however, be remembered that the loss of visual accommodation, and numbness and other sensory disorders, are particularly liable to be overlooked in children. I believe that in diphtheritic children, three at least out of every four are subsequently attacked by some form of paralysis.

(2) *Relation of the degree of paralysis to the quantity of the membrane.* It is no doubt true, as often stated, that some cases in which the membrane has been abundant escape, and that other cases in which the membrane has appeared in patches only, or in which it has been so slight and transient that the diagnosis was doubtful, are attacked by paralysis; but in my experience the cases in which the membrane was dense and widely distributed over the back of the throat were those in which paralysis most surely followed, especially in the general form in which the muscles of the extremities and trunk are implicated. Minor attacks of diphtheria are said to be particularly liable to be followed, especially in adults, by sudden paralysis of the heart; but I have seen four cases in which the membrane had been abundant, and in which death from syncope occurred after convalescence was fairly established.

(3) *Relation of paralysis to albumen in the urine.*—In almost all the severe cases of paralysis observed by me, albumen had been present in the urine, but this statement loses much of its value when it is considered that the presence of albuminuria is the most common reason for detaining a patient in the hospital after convalescence is otherwise established, and it is chiefly in patients so detained that an opportunity was afforded me of watching the onset and progress of paralysis. It is also possible that patients who had been discharged at an early period may have been attacked by paralysis after leaving the hospital, and it is, therefore, clear that my observations do not suffice to establish any definite connection between the presence of albuminuria and the subsequent development of paralysis. It does not appear to me that there is any definite proportion between the quantity of albumen present in

the urine, and the degree of the subsequent paralysis. In some cases of mild paralysis the urine had been loaded with albumen, while in cases of severe paralysis the quantity had only been small. In still other cases the paralysis had not been attended or preceded by any albumen in the urine, while in others there had been no paralysis although the urine had contained a large quantity of albumen.

(4) *Time at which the paralysis commences.*—The time at which the paralysis begins varies considerably. It sometimes begins before the disappearance of the membrane, and as early as at the end of seven days from the onset of the disease. In none of my cases has the first indication of paralysis been delayed beyond the fourth week of the disease, although new groups of muscles may be invaded at a much later period. The cardiac disturbance has, in nearly all my cases, appeared about the end of the third week.

(5) *The order in which and the relative frequency with which different muscular groups are invaded.*—The paralysis almost always begins in the muscles of the soft palate, or of the lower extremities, and these are attacked with about equal frequency amongst themselves, and far more frequently than any other muscular group. Then come successively in decreasing order of frequency, and increasing order of time of invasion, the internal and external muscles of the eyes, those of the upper extremities, the heart, the muscles of respiration, and last of all those of the face.

Soft palate.—Paralysis of the soft palate, shown by regurgitation of fluids through the nose, nasal quality of voice, and absence of movement on phonation, is nearly always symmetrical, but in one case the paralysis assumed a unilateral character. The regurgitation of fluids is generally intermittent, and the affection generally lasts rather more than a week.

The lower extremities.—Leaving the state of the knee-jerks out of account for the present, the first indication of the invasion of the lower extremities is usually afforded by distortions of the big toes. These distortions may be divided into three stages: (1) Hyper-extension at the proximal, and extension at the distal joint; (2) Hyper-extension at the proximal, and flexion at the distal joint; (3) Flexion at both joints. In some cases the three stages may be observed to follow one another in succession, but in other cases the third stage or position may be attained at once without being apparently preceded by one or other of the first two stages.

The eye muscles.—Next to the muscles of the soft palate and of the lower extremities, those of the eyes appear to be attacked with the greatest frequency. The paralysis may declare itself by loss of accommodation, or by double vision and squint, either separately or in combination, and it is difficult to say whether the internal or the external muscles are most frequently attacked.

The upper extremities.—The upper extremities become generally implicated a few days after the lower. Paralysis of the upper extremities generally declares itself, not so much by special implication of individual muscles as by a diffused feebleness of the limbs. The opponens pollicis, for example, has rarely been affected in a marked degree, and never except in association with great weakness of the arm as a whole.

Heart.—Implication of the heart is shown by attacks of syncope and of pseudo-angina. The first attack of syncope may prove fatal, or the patient may suffer from a succession of mild attacks, which end in recovery, or in a severe and fatal attack. The mild attacks seem to occur most frequently in adults, and severe and fatal attacks in children. The four fatal attacks which came under my observation all occurred in children under ten years of age, and in all the throat had been covered by a large quantity of membrane. In two of the adult cases observed the cardiac disorder consisted in a great increase in the frequency of the cardiac beats on slight exertion, such as sitting up in bed.

Respiratory muscles.—The intercostal muscles and the diaphragm may be attacked either separately or simultaneously, and in the latter event the disease generally proves quickly fatal. Implication of these muscles generally occurs at a late period in the course of the disease.

Facial muscles.—The muscles of the face were paralysed only in one case, and in that case the paralysis implicated the muscles of both sides, and the upper as well as the lower branches of the nerves.

Sensory disorders.—Sensory disorders appear to me to be much less common as a sequel to diphtheria than motor paralysis. Numbness and tingling of the finger tips and of the ulnar borders of the hands have been observed by me in several cases, but I have never seen anything approaching to absolute anæsthesia. The greater part of my observations, however, were made on children, in whom minor sensory disorders are not readily recognised, and it is, therefore, possible that the above conclusion is not applicable to adults. It is very probable that in all cases in which the reflex action of the palate was lost the surface was in a state of anæsthesia. In one case in which tracheotomy was performed the internal surface of the larynx and of a few of the upper rings of the trachea was insensible to the irritation caused by the passage of fluids through the glottis.

Duration of the paralysis.—The duration of the paralysis varies greatly in different cases. When it is limited to the soft palate, eyes, and a few of the muscles of the lower extremities, it may pass off in ten days or a week, or even in a shorter time. When it becomes generalised it may last for several months, but in that event a recovery takes place in one or more groups of muscles as other groups are being successively invaded.

Electrical reactions.—In the cases of paralysis implicating the extremities examined by me—ten cases in all—the faradic contractility of the affected nerves and muscles was diminished. In all these cases the galvanic reactions of the affected muscles, the KSc. was greater than, or equal to (in one case), the ASc., thus showing the qualitative change which characterises the reaction of degeneration.

It was found in three cases that the degenerative reaction was obtained in the quadriceps femoris before the patellar tendon reaction was quite lost, and in one case that the tendon reaction had reappeared before the normal formula was established in the muscle. The following seems to be the order of sequence :—

(1) Diminution of the patellar tendon reaction. (2) The partial reaction of degeneration in the quadriceps femoris. (3) Absence of the patellar tendon reaction. (4) Reappearance of patellar tendon reaction in slight degree. (5) Return of normal galvanic formula in the muscle and of normal degree of patellar tendon reaction.

Reflex actions.—The reflex of the palate is generally lost when the speech becomes decidedly nasal and fluids are ejected through the nose. The cremasteric, abdominal, epigastric, and plantar reflexes are occasionally diminished or absent.

The patellar tendon reaction is lost at an early period when the paralysis invades the lower extremities. In one case the patellar tendon reactions were decidedly exaggerated at first, but after a few days they became very feeble, although never entirely lost.

History.—Records of cases of paralysis after diphtheria might be multiplied, but the few here reported sufficiently illustrate the course and progress of the disease. Such striking phenomena as are presented in diphtheritic paralysis must have attracted attention in all ages, but it is possible that the difficulties in speaking and swallowing may have been regarded as having resulted from a local cause, and the paralysis of the trunk and extremities as resulting from general weakness. In any case, it seems certain that the connection between the antecedent throat affection and the subsequent paralysis had not been clearly made out until about the middle of last century. It is true that a passage from Severinus[*] is often quoted to show that this observer had recognised the connection, but it simply states that sudden death may occur from thirty to forty days after convalescence had been established.

The first observation of paralysis of the soft palate appears to have been made by Chomel.[†] Of one patient, a girl, aged six and a half years, he says "she had become truly convalescent only on the forty-fifth day of the disease, inasmuch as up to that time she expressed herself with difficulty, and spoke through the nose, while the soft palate was trailing." And of another young patient he says, "I have learned that up to the fortieth day of the disease the patient spoke through her nose, and that she became squint-eyed and disfigured, but as her strength gradually returned her aspect became natural." About the same time Ghisi,[1] in describing the epidemic of angina which appeared in Cremona in 1747 and 1748, remarks of his own son, who had suffered from a severe attack of the disease: "We abandoned to nature the task of remedying the strange effects of this disease, effects which were observed in many of those who had already become convalescent, and which persisted for about a month after the disappearance of the angina and abscess. The child continued to speak through his nose; and the food, chiefly fluids, instead of passing into the œsophagus, frequently returned through the nose." A few years later, Fothergill[2] wrote: "Those who survived the fourteenth day were thought to be out of danger, at least from the disease itself, though some dropped off unexpectedly after a much longer reprieve. The consequences of this disease were often felt a long time after it had ceased; an excessive langour and weakness continued for many months, and the voice or deglutition was frequently affected so as to be perceivable in some almost a year after."

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[*] "Marci Aurelii Severini de recondita abscessum natura, libri VIII." Francofurti ad Moenum, 1643, p. 440.

[†] Chomel. "Dissertation historique sur l'éspèce de gorge gangréneux qui à regné parmi les enfans l'année dernière." Paris, 1749, pp. 33 et 35.

An American physician, Dr. Samuel Bard,[3] published an account of an epidemic of suffocative angina which appeared in New York in 1771, and mentioned the case of a child, aged 2½ years, who had suffered from paralysis of the soft palate and partial paraplegia after an attack of sore throat. In the early part of this century brief reference is made by several French authors[4] to the disorders of sight and of speech and the weakness of the extremities which follow an attack of diphtheria; but it was not until 1851 that a general impulse was given to the study of these symptoms by publication of the observations of Trousseau and Lassègue[5] and of Morisseau,[6] all of whom at that time believed that the paralysis was caused by a local inflammation. In the earlier Memoirs of Bretonneau no mention appears to be made of paralysis as a sequel of diphtheria, and it was not until 1855 that he published the case of Dr. Herpin,[1] although as early as 1843 that physician had sent him a report of his symptoms. After describing the symptoms of the acute stage, Dr. Herpin proceeds:—"Incomplete recovery, paleness; a fortnight later there was pain in the wrists; confusion of sight; constriction of the throat; paralysis of the palatine vault, which had become completely insensible; regurgitation of food by the nostrils. Rather later there was tingling in the great toes, ascending as far as the knees. I walked with difficulty and very slowly, and my weakness was especially painful when I went up stairs, and this state continued without improvement for six weeks. The same tingling had reached my hands and fingers, and I had completely lost all tactile power." In the same memoir Bretonneau[2] mentions the case of a robust boy, aged 12 years, who was brought to him three months after he had recovered from a severe attack of diphtheria, "walking by himself, but looking at his feet to know if, after three months, they touched the ground. They still remained so destitute of tactile power that he appeared to himself to be walking in the air." Important contributions now followed one another in France in quick succession, the most noteworthy of these being the papers of Trousseau,[3] Farre,[4] Bouillon-Lagrange,[5] Moynier[6]; the inaugural dissertations of Peraté,[7] Péry,[8] Boutin,[9] Ranque,[10] Revillond,[11] Espagne,[12] and, above all, the exhaustive monograph of Maingault,[13] and the brilliant chapter on the subject in Trousseau's[14] "Clinical Medicine."

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[1] Ghisi. "Lett. medich. ist. delle angina epidem. degli anni 1747 et 1748." Quoted by Bretonneau (P.). "Des inflammations spéciales du tissu muqueux, et en particulier de la diphthérite, ou inflammation pelliculaire, connue sous le nom de croup, d'angine maligne, d'angine gangréneuse, etc." Paris, 1826, p. 460.

[2] Fothergill (John). "An account of the sore throat attended with ulcers." 3rd edition. London, 1751, p. 17.

[3] Bard (Samuel). "An enquiry into the nature, cause, and cure of the angina suffocativa, or sore-throat distemper."—*Transactions of the American Philosophical Society*, Vol. I., Philadelphia, 1789, p. 388.

[4] Guimier. "Mémoire sur une épidémié d'angine maligne, ou diphthéritique, qui a regné à Vouvray et dans les communes voisines à la fin de 1826 et dans le córant de 1827.—*Journal génér. de Méd.*, Tome CIV., année 1828. Et Ozanam (J. A. J.). "Histoire médicale générale et en particulière des maladies épidémiques." Seconde Edition, Tome III., 1835.

[5] Trousseau (A.) et Lassègue (Ch.). "Du nasonnement de la paralysie du voile du palais." —*L'Union Médicale*, 1851, p. 471.

[6] Morisseau (L.). "Paralysie du voile du palais comme cause du nasonnement."—*L'Union Médicale*, Paris, 1851, p. 499.

At a later period the contributions of Charcot and Vulpian[15], Roger[16],

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[1] Bretonneau. "On the means of preventing the development and progress of diphtheria." Fifth Memoir.—*Arch. gén. de Med.*, January and September, 1855. "Memoirs on Diphtheria,"—*New Syd. Soc.*, Lond., 1859, p. 181.

[2] *Op. cit.*, p. 203.

[3] Trousseau (A.). "Des angines."—*Gaz. des Hôpitaux*, July, 1855.

[4] Farre. "Des accidents consécutifs de la diphthérie."—*L'Union Médicale*, 1857, p. 57.

[5] Bouillon-Lagrange. "Quelques rémarques sur l'angine couenneuse épidémique, 1857-1858.—*Gaz. hebdomadaire*, June 24, 1859.

[6] Moynier. "Compte rendu des faits de diphthérie observés pendant le premier sémestre de l'année 1859.—*Gaz. des Hôp.*, 1859, pp. 497, 507, 529 and 542.

[7] Peraté. "Sur la diphthérie."—*Thèse de Paris*, 1858.

[8] Péry,[9] Boutin,[10] Ranque,[11] Revillond.—*Thèses de Paris*, 1859.

[12] Espagne.—*Thèse de Paris*, 1860, and *Gaz. des Hôp.*, Tome XXXIII., Paris, 1860, p. 77.

[13] Maingault (A.). "Sur les paralysies diphthériques."—*Arch. géner. de Méd.*, Tome XIV., Vol. II., Paris, 1859, pp. 385 et 674.

[14] Trousseau (A.). *Clinique médicale de l'Hôtel-Dieu de Paris*, Tome I., Paris, 1861, p. 370.

[15] Charcot et Vulpian.—*Comptes render de la Soc. de Biol.*, 1862, et *Gaz. Hebdomadaire*, 1862.

[16] Roger (H.). "Researches cliniques sur la paralyse consécutive à la diphthérite."—*Arch. génér. de Med.*, 5th Série, Tome XIX., Paris, 1862, pp. 5, 199 et 460.

Roger and Peter[1], Lorain and Lépine[2], Gendron[3], Pierret[4], Vulpian[5], Déjerine[6], Landouzy[7], and others, added so much to our knowledge of the disease that French physicians may well claim the distinction of having taken a predominating share in depicting accurately the clinical history, and determining the pathology of this form of paralysis.

The first important contribution to the study of diphtheritic paralysis which appeared in Germany was as late as 1862, from the pen of Dr. Hermann Weber[8], who was at the time physician to the German Hospital in London, but it was soon followed by papers from Max Jauffé[9], Buhl[10], Gerhardt[11], Meyer[12], and others.

In this country the first observation of paralysis following an attack of diphtheria since the days of Fothergill appears to have been made (in 1858) by the late Sir W. Gull.[13] The patient was a boy who had suffered from an attack of diphtheria which lasted about a fortnight, and after being convalescent for three weeks he noticed that he did not carry his head erect; then followed difficulty of deglutition, loss of voice, dyspnœa, and at the end of two days deglutition became impossible, the breathing became thoracic owing to paralysis of the diaphragm, and the patient died from asphyxia. The muscles of the neck were flaccid and wasted, the arms were unaffected. There was tenderness to pressure over the transverse processes of the upper cervical vertebræ. He mentions another case in which the arms were paralysed, but without giving the symptoms in detail. Sir W. Gull attributed the paralysis in this case to a myelitis of the upper part of the spinal cord, secondary to an ascending neuritis of the cords of the cervical plexus, which was in its turn caused by a chain of inflamed glands.[1] This opinion was in accord with the theory he had already advanced, that myelitis of the lumbar enlargement of the cord is often a result of an ascending neuritis secondary to a local disease in the uro-genital organs.

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[1] ROGER et PETER. Art. "Angine diphthérique."—*Dict. Encyclop. des Sciences médicales*, Série I., Tome V., 1866, p. 32.

[2] LORAIN et LÉPINE. Art. "Diphtherie."—*Nouveau Dict. de Méd. et de Chir.*, Tome II, 1869, p. 619.

[3] GENDRON. "De la dysphagie diphthéritique."—*L'Union médicale*. Série II., Tome V., 1866, p. 569.

[4] PIERRET.—*Société de Biologie*, Dec. 23, 1876.

[5] VULPIAN. "Maladies des système nerveux." Paris, 1876.

[6] DÉJERINE. "Note sur l'existence des lesions des racines anterieur dans la paralysie diphthéritique,"—*Gaz. méd. de Paris*, Série IV., Tome VI., 1877, p. 464. "Recherches sur les lésions du systéme nerveux dans la paralysie diphthéritique."—*Arch. de Physiologie*, Série II., Tome V., 1878, p. 107, et "Sur les lésions der systéme nerveux dans cinq cas de paralysie diphthéritique."—*Gaz. Hebdom de Méd.*, 1877, p. 814.

[7] LANDOUZY (LOUIS). "Des paralysies dans les maladies aigues." Paris 1880, p. 32, *et seq.*

[8] WEBER (HERMANN). "Ueber die Lähmungen nach Diphtheria."—*Virchow's Archiv*, Bd. XXV., Berl., 1862, p. 114, und Bd. XXVIII., 1863, p. 489.

[9] MAX JAFFÉ.—*Schmidt's Jahrb.*, 1862, Bd. 140, und 1868, p. 215.

[10] BUHL. "Einiger über Diphtheria."—*Zeitschrift für Biologie*, 1868.

[11] GERHARDT. "Diphtheritis; Lähmung des Kehlkopfschluss-apparates; fortbestende Diphtheritis des Sinus pyriformis; Schluckpneumonie; Heilung durch Faradisation des Kehlkopfes und ausschliessliche Ernährung durch die Schlundsonde während 32 Tagen."—*Berl. klin. Wochenschrift*, Bd. VI., 1869, p. 44.

[12] MEYER (P.). "Anatomische Untersuchungen über diphtheritische Lähmung."—*Virchow's Archiv.*, Bd. LXXXV., 1881, p. 196 *et seq.*

[13] GULL (W.) "Lesions of the nerves in the neck and of the cervical segments in the cord after 'faucial diphtheria.'"—*The Lancet*, Vol. II., 1858, p. 4.

An epidemic of diphtheria broke out at Cornwall in 1855, and in the following year it appeared at Folkestone in Kent, Spalding in Lincolnshire, and Leek in Staffordshire; and when once Sir W. Gull had directed attention to paralysis as a sequel of it, reports of cases came from various parts of the country. Cases of paralysis were reported by Kingsford[2], from Clapton; by Bellyse[3], from Nantwich, in Cheshire; by Hugh George[4], from Revesby, in Lincolnshire; by Ranking[5] and Eade[6], from Norwich; by Ransome[7], from Bowdon, in Cheshire; by Hillier[8], from Plymouth, and by Ellis[9] from Crowle, in Lincolnshire; while Dixon[10] and Rooke[11] directed special attention to the impairment of vision which results from paralysis of the accommodation. Our knowledge of this subject was still further extended and perfected by the various communications and the monograph of Greenhow[12], the able articles which appeared in the second report of the Medical Officer of the Privy Council[13], and by the monograph of Hart[14], and the classical work of Jenner[15]. The appended list of contributions indicates the sources of our further knowledge of the disease :—

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[1] Gull (W.). "Cases of paraplegia associated with gonorrhœa and stricture of the urethra."—*Medico-Chirurgical Transactions*, Vol. XXXIX., Lond., 1856, p. 195.

[2] Kingsford (Chas. D.). "Letter on 'Diphtheria.'"—*The Lancet*, Vol. II., 1858, p. 485.

[3] Bellyse (E. S.). "Letter on 'Diphtheria.'"—*The Lancet*, Vol. II., 1858, p. 573.

[4] George (Hugh). "Letter on 'Diphtheria.'"—*The Lancet*, Vol. II., 1858, p. 619.

[5] Ranking (W. H.). "A lecture on diphtheria, delivered at the Norfolk and Norwich Hospital."—*The Lancet*, Vol. I., 1859, pp. 28 and 51.

[6] Eade (Peter). "Cases of paralysis as a sequela of diphtheria."—*The Lancet*, Vol. II., 1859, p. 56.

[7] Ransome (A.). "Diphtheria affecting the throat, and, to a slight extent, the air passages: paralysis; recovery."—*The British Medical Journal*, Vol. II., 1859, p. 906.

[8] Hillier (Thomas). "On Diphtherite."—*Medical Times and Gazette*, Vol. I., 1859, p. 135.

[9] Ellis (Henry W. T.). "Diphtheria at Crowle, in Lincolnshire."—*The Lancet*, Vol. II., 1859, p. 642.

[10] Dixon. "Impaired vision as a sequela of diphtheria."—*The Medical Times and Gazette*, Vol. I., 1859, p. 389.

[11] Rooke (T. M.). "A letter 'on impaired vision after diphtheria.'"—*The Medical Times and Gazette*, Vol. I., 1859, p. 659.

[12] Greenhow (Headlam). "Communication, Harveian Society."—*The Medical Times and Gazette*, Vol. II., 1859, p. 294; and on "Diphtheria." London: 1859 and 1860.

[13] Simon, Greenhow, Sanderson, Gull, Monckton, Moyce, and Jones. Articles in the Second Report of the Medical Officer of the Privy Council, with appendix (1859). Lond., 1860.

[14] Hart (Ernest). "On Diphtheria: Its symptoms, treatment, and prevention." London: 1859.

[15] Jenner (William). "Diphtheria: Its symptoms and treatment." London: 1861.

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Etiology.—Although an attack of diphtheria is always the exciting cause of the subsequent paralysis, yet it is likely that the relative frequency with which patients are affected by paralysis will be greatly influenced by the age and sex and previous state of health of the patient.

Age.—From an analysis of his cases Landouzy* concludes that diphtheria is more frequently followed by paralysis in adults and advanced age than in infancy. Out of 68 cases collected by him, 6 only occurred between the ages of 2 and 6; 9 between 6 and 10; 18 between 10 and 20; 13 between 20 and 30; 8 between 30 and 40; 8 between 40 and 50; and 6 above 50 years of age. Now, according to the statistics of Trousseau, Rilliet and Barthez, and others, diphtheria is encountered much more frequently in infancy, or from the ages of 2 to 6 years, than at any subsequent period of life, and it may be said that there is a progressive decline in the relative number attacked in each decade of life, until after 45 years of age the proportion of the population affected becomes exceedingly small. But the statistics of Landouzy show that 15 only are attacked by paralysis in the first, as compared with 18 in the second, 13 in the third, 8 in the fourth, and 6 in the sixth and subsequent decades of life; so that taking into consideration the relative frequency of diphtheria in early, and its relative infrequency in later life, it would seem that paralysis must follow diptheria much more frequently in adults and old people than in children. An analysis of my own collected cases, however, would appear to throw considerable doubt upon this conclusion. The number of cases collected by me from the sources mentioned in the foregoing bibliography is 171; of these 38 occurred in patients between the ages of early infancy and 6 years; 25 between 6 and 10; 42 between 10 and 20; 31 between 20 and 30; 23 between 30 and 40; 6 between 40 and 50; and 6 above 50 years. In other words, of these 171 cases, 63 occurred in the first, 42 in the second, 31 in the third, 23 in the fourth, 6 in the fifth, and 6 in the sixth and subsequent decades of life. These statistics, then, lend much less countenance than those of Landouzy to the view that paralysis follows diphtheria less frequently in children than in adults; and, besides, it must be remembered that a moderate degree of paralysis is much more liable to be overlooked in infants than in adults. An infant cannot direct attention to numbness, tingling, and other sensory disorders, and inability to see small objects from paralysis of accommodation, and a moderate squint, is apt to be entirely overlooked by unobservant parents, while incapacity to stand or walk is likely to be attributed to general weakness instead of to a special kind of paralysis. And, again, a much larger proportion of children than of adults die during the acute stage of the disease, and before the period for the appearance of the paralysis is reached. On the whole, it seems probable an extended observation will show that diphtheria is followed by paralysis as frequently in children as in adults.

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* Landouzy, *Op. cit.*, p. 42.

Sex.—Of 93 cases collected by Landouzy, 55 males and 38 females were attacked by paralysis, and of my 171 cases, 91 were males and 67 females, while in 13 cases the sex is not stated. The statistics of writers on diphtheria seem to show that males are rather more frequently attacked by diphtheria than females, and it would, therefore, seem probable that sex does not exercise any influence upon the development of paralysis after diphtheria.

Relation of the Sore Throat to the Subsequent Paralysis.—A survey of my collected cases has not enabled me to establish any proportion between the severity of the sore throat and the degree and extent of paralysis, but it is abundantly clear that many cases of slight sore throat are followed by well marked paralysis. Reverting to the cases recorded by myself, it will be seen that the first patient (Case 1) had suffered from a very severe attack of diphtheria, which appeared likely at one time to have proved fatal, but the second patient (Case 2) was only supposed to have had a common inflammatory sore throat, until the appearance of the paralysis, and, indeed, the opinion that the sore throat was of the nature of diphtheria, is only an inference from the presence of subsequent paralysis. The third patient (Case 3)—a medical man—on being asked if he had recently suffered from sore throat, replied in the negative, and it was not until he was told that his symptoms bore a strong resemblance to diphtheritic paralysis, that he mentioned the circumstance of having attended several cases of diphtheria in his practice, while the fact of his having had a slight sore throat himself only came to his recollection some days later. Some time ago, another medical man came to me complaining of numbness of the extremities, and general weakness. He stated that some weeks previously he experienced disagreeable sensations of numbness, and tingling in his fingers and toes, but he did not feel alarmed about himself until one day he found, on joining at a game of cricket, that he could not run. When I saw him he had lost all his spring, could not stand on the tips of his toes, the patellar tendon reactions were absent, his grasp was feeble, and he had some difficulty in performing the special movements of his fingers; but his speech, power of deglutition, and vision were unaffected. All the causes of peripheral neuritis, including sore throat, were strictly enquired into with negative results, and the patient left me with a hopeful prognosis, and the diagnosis of idiopathic multiple neuritis. Some weeks later he wrote to say that he had completely recovered, and that he believed his case must have been one of diphtheritic paralysis, as he had been attending cases of diphtheria, and he now recollected having had a slight sore throat some weeks before he saw me.

A fortnight ago (October 2nd) a professional brother, who has the medical superintendence of a large educational establishment in the neighbourhood of Manchester, consulted me for paralysis of all the extremities. He could hardly stand, and had to be assisted from the cab to my consulting room. The patellar tendon reactions were absent, and, although he is a powerfully-built man, the grasp of either hand only measured 20 degrees on the dynamometer, as compared with 100 degrees of a healthy man. There was possibly a slightly nasal quality of speech, but the uvula was not trailing, and it contracted on phonation and to mechanical irritation, and there were no eye symptoms. Referring to his then condition, he said: "This has a history. In the beginning of March I had a severe attack of influenza, which left me very weak for two or three months, but after a time I picked up, and felt quite well during the months of June, July, and the early part of August. About the second week in August I felt again a good deal run down, but at the middle of the month I got off for my holidays, went up the Rhine, spent ten days in Switzerland, and came back on September 1st to London, feeling quite well. In London I took a Turkish bath, and spent the remainder of a rather cold day boating on the river. On the following day I came home, and for the next two days I felt as well as ever I did in my life. On the third day my hands and feet began to tingle and became numb, and I soon found that I was becoming day by day weaker, until I am as you see me now, scarcely able to stand." He then added: "You may put alcohol and syphilis out of the question in my case." "Have you had a sore throat recently?" I asked. "No," he replied, unhesitatingly. "Just think," I said, "when you were run down in the second week in August; are you quite sure that you had no sore throat?" "Not the least," he replied; "I have not had a sore throat this year." "Well," I said, "if you are not suffering from diphtheritic paralysis, I do not know what to make of your case." "Oh!" he replied, "we have had a lot of diphtheria at our place, and during August all the drains were opened and I had to superintend them." He still persisted, however, in saying that during August he had not the slightest indication of a sore throat, and that he had nowhere an open wound. Only this very day (October 17th) a man came to my consulting rooms complaining of numbness and coldness of the extremities, and saying that his feet felt as heavy as if they were made of lead. The patellar tendon reactions were absent, and there were indications of a slight degree of paralysis of the small muscles of the hands and feet. A brief examination sufficed to exclude the action of lead, alcohol, and syphilis, and I then enquired if he had recently suffered from sore throat. "I had an attack of quinsy in August," he said, "and in the following week one of my children died from croup. The doctor opened the throat and put a tube in [tracheotomy], but the child died two days afterwards." I then found on enquiry that in the week subsequent to the child's death he had lost his voice, although his throat was no longer sore, and some days later he had double vision, and was unable to read without the aid of glasses. The visual disorders lasted a week or ten days, and then his fingers and toes began to tingle, and his legs soon became so heavy that he could scarcely walk. A case came before me about Christmas, last year, in which the relation of the paralysis to an antecedent sore throat was involved in still greater obscurity. The patient was a farmer, and I was told that he had not been able to work since he had an attack of rheumatic fever eighteen months previously. His chief complaint was that he could not work with a spade or other tool, owing to the feebleness of his grasp. He said that his legs were at one time so weak that he could hardly stand, but he had now completely recovered the use and power of his lower extremities. His hands and feet were for a long time quite numb and dead, and without feeling, but the natural feeling had now returned, with the exception of a slight impairment of the sense of touch in the tips of his fingers, which was still present. I found the patellar tendon reactions present, and the only objective symptoms I could discover were a much feebler grasp than might have been expected from a man accustomed to manual labour, and a certain disability in performing the special movements of the fingers. There was no indication of syphilis in the history of the patient, he was a life long total abstainer, and there was no blue line in the gums. I was inclined to regard the case as one of multiple neuritis of rheumatic origin. It occurred to me, however, to make enquiries into the kind of symptoms from which he suffered during his supposed attack of rheumatic fever. "Had you a sore throat," I asked, "when you began with the rheumatic fever?" "Oh! yes," he replied, "that is the way I began. I went to a doctor and he looked down my throat; he told me I must go to bed at once. He came to see me every day for over a week, and he applied stuff to my throat with a brush, and he gave me a gargle and medicine." He had difficulty in swallowing while in bed, but he could not remember that he had much difficulty at a later period, or that he had double vision, or difficulty in reading. A few weeks later his hands and feet became numb; "in fact," he said, "I was numb all over, and I could scarcely stand, whilst things slipped out of my hands, and I could hardly button my clothes." After this statement it became quite clear to me that I had in the case before me the last remnants of a paralysis that resulted from a diphtheritic sore throat, from which the patient had suffered eighteen months previously. Our search after diphtheria, as an antecedent to peripheral neutritis, is obscured in various ways. In the following case the symptoms were supposed to have resulted from an injury to the head, but on careful inquiry it turned out that the paralytic phenomena had appeared soon after the patient had suffered from sore throat, and many months after the accident. P. H., aged 35 years, was brought to the Royal Infirmary to see me in March last, with the statement that he was suffering from paralysis, which was caused by an injury to the head received in May, 1889. When engaged at work a brick fell on the top of his head and knocked him down. There was a gaping wound on the top of the head, and he lost a great quantity of blood, but did not lose consciousness. On examining the patient, a cicatrix, an inch in length, was found on the top of the head above the right parietal eminence, and about an inch to the right of the middle line. The neighbourhood of the cicatrix was tender to pressure, but the bone was not depressed, and at no time did he feel any indication of spasm or convulsion. He complained of sleeplessness, depression of spirits, loss of memory, giddiness, and occipital headache. The field of vision of the right eye was much restricted, and of the left normal; there was relative deafness of the right ear, and the prick of a pin was not felt so well in the right half of the head, trunk, and extremities, as in the corresponding parts of the left half of the body. He also complained of great weakness, and his gait was unsteady, and had a well-marked "high stepping" character, and the patellar tendon reactions were absent. The cutaneous reflexes were active. The grasp of either hand was feeble, and he could not approximate the tips of the thumbs and little fingers without flexing the thumbs at the phalangeal joints. There were no sensory disorders to objective examination, beyond the slight degree of heminanæsthesia already described; but the patient stated that some time ago his hands and feet were numb, cold, and without feeling. The pupils were somewhat dilated, but contracted to light and accommodation. The ocular movements were normal, with the exception of outward rotation of the left eye, which was slightly defective, as proved by the inability of the patient to bring the outer edge of the cornea quite up to the outer canthus. The electrical reactions of the affected nerves and muscles were normal. The urine was acid, specific gravity 1030, and was free from sugar or albumen. It was clear that in this case we had to do with two sets of quite distinct symptoms. The insomnia, depression of spirits, occipital headache and right sided hemianæsthesia, with restriction of the field of vision, were clearly the symptoms of traumatic hysteria, as so frequently met with after railway accidents. The fact that the hemianæsthesia occupied the right half of the body showed clearly that it could not have resulted directly from the injury, which was seated over the right parietal eminence. The paralysis was too equally distributed on both sides of the body and in all the extremities to be attributed to the local injury directly, and besides, the loss of the patellar tendon reactions showed that we had to do with an atrophic paralysis; in short, taking the history of the numbness of the extremities, from which the patient had previously suffered, together with the character of the paralysis, there could be little doubt that the patient was suffering from a peripheral neuritis. The question now was, how to account for the presence of multiple neuritis? The patient was a total abstainer; there was no history of syphilis; there was no blue line on the gums; and the urine was free from sugar; while the patient was far too powerfully built and healthy looking to attribute his condition to any form of cachexia; and he was not engaged in using explosives, or in any occupation that would expose him to carbon monoxide gas. A careful search into the history of the onset of his symptoms was now instituted. After the injury he bled freely, and was nine days in bed. On getting up he felt very feeble, but at the end of three weeks he resumed his occupation, and although he still felt weak and depressed he continued at his work for three months. At the end of that time—that is, about five months before he came under my observation—he was seized with a sore throat, and had to desist from work. He was about a week in bed, and had some difficulty in swallowing at the time, but not at a subsequent period. On getting up he felt very weak, and a fortnight later he began to see things double, and was troubled by attacks of giddiness. Soon afterwards his hands and feet became numb and tingled, and his legs got so weak that he could hardly stand, while he could not hold any tools in his hands. During the few weeks before I saw him he had improved considerably in his power of walking, and his grasp was getting stronger, while the numbness had disappeared from his extremities, but he still complained of great weakness and depression, and was quite unable to work. Comment on the case is unnecessary. I have no doubt that the symptoms indicated a mixture of traumatic hysteria and post diphtheritic paralysis.

In other cases the presence of diphtheria is obscured by an antecedent attack of tonsillitis. A case is mentioned by Dr. Buzzard, in which a patient immediately after the breaking of a "quinsy," a disease from which he had suffered on eight or nine previous occasions, went down to a cellar in his house in which there was an untrapped drain, which stank offensively, and a few days later he began to feel numbness of his feet, and was subsequently attacked by paralysis, which presented all the characteristics of that which follows diphtheria. Of this case Dr. Buzzard* remarks, "it would seem that with a tonsil presenting an open wound he became readily infected by this exposure." At other times the difficulty in diagnosis arises from the presence of an antecedent inflammatory sore throat or tonsilllitis, suggesting that the paralysis is due to diphtheria, when in reality the sore throat has only helped to develop, as any other acute illness might have done, a paralysis from a totally different cause, such as excessive drinking. A case of this kind is recorded by Dr. Bristowe†, in which a policeman developed an atrophic paralysis immediately after he had suffered from a severe attack of tonsillitis, but the previous habits of the patient, and the presence of great muscular tenderness, extreme atrophy of the muscles, and persistence of the paralysis with deformities of the extremities, after being under careful treatment for nine months, pointed to alcohol rather than diphtheria as the cause of the symptoms. Another difficulty in diagnosis arises from the fact that paralysis may follow cutaneous diphtheria, and in the complete absence of any throat affection. Cases of this kind have been reported by Roger, Raciborsky, Paterson, Caspary, and Garnier, and when we come to speak of the cases of paralysis which are said to have followed septicæmia, evidence will be adduced to render it probable that in some of theses cases, at least, the wound had been infected by the poison of diphtheria. In commenting upon the principal case of acute ascending paralysis, reported by Landry, it has already been suggested in these pages that the paralysis was a sequel to infection of the ulcerated surface left by the blisters used in the treatment of an antecedent pneumonia, and when we come to analyse the other cases of paralysis reported as occurring after pneumonia, still further evidence will be adduced to show that the patients had been infected by diphtheria on a blistered surface, or that the attack of diphtheria was obscured by a complicating pneumonia. In estimating the evidence in favour of the occurrence of paralysis after scarlet fever, typhoid fever, small-pox, and erysipelas, the possibility of a complication of diphtheria with the chief disease must not be overlooked.

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* BUZZARD (THOMAS). "Clinical lectures on diseases of the nervous system."—London, 1882, p. 317.

† BRISTOWE (J. SYER). "Clinical lectures and essays on diseases of the nervous system."—London, 1888, p. 355.

The remarkable difficulties which occasionally present themselves in connecting a rapidly invading paralysis with the poison of diphtheria are well illustrated by the history of an epidemic of diphtheria as reported by Dr. Boissarie,* which occurred in one of the departments of France. The first victim to the disease was a child, aged three years, who was seized, without any antecedent sore throat, by difficulties in deglutition and articulation; the breathing soon became embarrassed, and the child died in four or five hours from the commencement. The mother, who had been confined two months previously, was next attacked in the same way, and in addition to paralysis of the pharynx and tongue, the upper eyelids were drooped, the pulse became very quick, and the breathing slow and embarrassed, and she died in twenty-four hours. The grandmother and son and another daughter were successively attacked, and each died in six, five, and three days respectively; so that of a family of six persons, five died, and the husband of the woman who had been confined alone escaped. An officer in a neighbouring garrison was next attacked. On October 15, the soft palate and pharyngeal muscles became suddenly paralysed, and three days later he brought up large quantities of mucus from the mouth; his face was altered, his eyes were half closed, and he had dimness of vision. On October 25th, he complained for the first time of sore throat, and the uvula and fauces were now seen to be covered by a vast exudation, which for five days rendered swallowing impossible. He also experienced difficulty in using his arms, and his pulse was slow. This patient recovered. A young woman, who had on October 21st visited the family first affected, found herself on October 25th, without any pain or visible disease of the throat, unable to swallow liquids. On the following day her eyelids were drooped, and her vision was dim. A few days later her breathing became embarrassed; the pulse was small, feeble, and beat at 80 in the minute; the urine passed involuntarily, and the patient died, without agony, from simultaneous arrest of respiration and of the heart's action, on Oct. 30th, five days from the commencement of the symptoms. A child of this woman, of the age of 10 years, was seized with the same symptoms as her mother, and was also unable to stand, but she made a complete recovery in about two months. The porter of the hospital was next attacked by paralysis, without antecedent sore throat; his gait was uncertain and staggering, his arms were feeble, and he was unable to use his fingers in buttoning his clothes, but he made a complete recovery in three months. Dr. Boissarie was now himself attacked; he began on Oct. 31st, with a painful sensation of a foreign body in his throat, but it was free from membrane. He could not swallow without taking great precautions, and his voice was nasal. The urine contained albumen, his pulse, which was very feeble, fell to 50 beats in the minute, and his vision was dim, but he made a complete recovery in two months. Here, then, is an epidemic which was ushered in by cases in which the paralysis occurred at once, without being preceded by angina, and proved fatal in a few hours or days. In one case the paralysis was followed by angina instead of the angina preceding it. In the further progress of the epidemic the disease followed the usual course, in which the angina was the predominant symptom, and in most of the cases it was neither preceded nor followed by paralysis.

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* BOISSARIE. "Epidémie de paralysies diphthéritiques."—*Gaz. hebdomadaire*, Série II. Tome XVIII. Paris, 1881; p. 310.

Relation of Albuminuria to the Degree of Paralysis.—The presence of albumen in the urine does not appear to exercise any appreciable influence upon the development of the subsequent paralysis. In my 171 collected cases albumen in the urine is only mentioned 10 times. Of these 10 cases 2 died—1 at the end of 38 and the other at the end of 36 days from the onset of the sore throat—and 8 recovered in a period varying from 24 days to 4 months from the onset of the sore throat, the average duration being 2 months. In a large number of my cases, however, the symptoms of the primary disease are merely recorded from the statements of the patients and their friends, and the albuminuria is mentioned chiefly when it is present during the course of the paralysis. My cases, therefore, do not enable me to determine whether or not there is any relation between the presence of albumen in the urine during the acute stage and the development of paralysis subsequently.

Relative Frequency of Paralysis after Diphtheria.—The frequency with which diphtheria is followed by paralysis has been variously estimated by different authors. Out of 210 cases of diphtheria collected in 1860 by Roger* 36 were subsequently affected by paralysis, which gives a proportion of rather more than 17 per cent, but in a subsequent communication the author* stated his belief that the proportion affected was still higher.

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* ROGER. "Récherches cliniques sur la paralysie consécutive à la diphthérite."—*Arch. génér. Méd.*, Série V., Tome XIX., 1862, p. 9.

Sanné collected 1,382 cases, and of these 15·5 were subsequently affected by paralysis, which gives a proportion of about 11 per cent; while Dr. Monckton, of Maidstone, only met with paralysis nine times in 300 cases, or a proportion of 1·15 per cent. It will be remembered that the number of cases of diphtheria treated at Monsall Fever Hospital in the period over which Dr. Bowman's observations extended was 90, and that of these 20 were subsequently affected by paralysis, which gives a proportion of 22 per cent. But if account be taken of the cases in which death occurs at an early stage of the primary disease, and of the patients that disappear from observation before the time for development of the paralysis is reached, it is clear that the proportion attacked will be much higher than that indicated by these statistics. And when, by close observation, the slighter indications of paralysis—such as a nasal quality of voice, dimness of vision, diplopia or strabismus, loss of the patellar tendon reactions, and slight deformities in the attitudes of the toes and fingers—are carefully noted, it is very probable Mr. Bowman's estimate that two out of every three children affected by diphtheria are subsequently attacked by paralysis, or a proportion of 66 per cent, is not by any means too high. It is, however, possible that the proportion attacked by paralysis varies greatly in different epidemics; but in the meantime, taking one epidemic with another, we do not think that the estimate of 20 per cent is by any means too high, and the probability is that a careful observation of the slighter degrees of paralysis will hereafter show that this estimate is much below the reality.

Symptoms.—It has already been said that no definite relation could be discovered in my cases between the duration and severity of the primary disease and the extent and degree of the subsequent paralysis. Of the 171 cases collected by me no information is given with regard to the duration of the primary disease in 42 cases; the primary sore throat is described as having only lasted a few days, or having been of short duration, or slight in degree in nine cases, while it is said to have been severe in five cases. Of the remaining 115 cases, the sore throat is said to have lasted in 15 cases from 3 to 7 days, in 80 cases from 8 to 14, in 15 cases from 15 to 21, and in 5 cases from 22 to 28 days. It is not likely that the membrane lasted in any case so long as 22 days, but it may be assumed that in those cases in which it is mentioned that convalescence was not fully established until the course of the fourth week, the primary disease must have been of a very severe character;

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* Roger et Peter. Art. "Angine diphthéritique."—*Dict. Encyclop. des Sciences Médicales*, Série I., Tome V., 1866, p. 32.

yet in none of those cases was the subsequent paralysis profound in degree, while in many of the cases in which the sore throat was slight and transient, the paralysis was widely distributed, and in some of the cases proved fatal. With regard to the interval between the cessation of the sore throat and the commencement of the paralysis, in 43 cases, no information is given; while in 13 cases the paralysis is said to have begun at the close of the febrile attack. The case of John Dunlop (Case 1) reported by myself is instructive in this respect. It will be seen that during the course of the sore throat, his voice became husky, and fluids regurgitated through the nose; but as improvement of the local disease set in, these symptoms all but disappeared, and remained absent until the fourteenth day after convalescence had become established, when the nasal voice and regurgitation of fluids through the nose reappeared, and lasted for about three weeks. In one of the cases reported by Déjerine, the patient, a child, aged 2½ years, who was the subject of Potts's curvature, had a slight attack of angina, which lasted thirteen days, and towards the end of the attack he had some difficulty in swallowing fluids, part of which regurgitated through the nose. At the end of three days all signs of paralysis of the veil of the palate had completely disappeared, and the little patient was regarded as quite convalescent; but at the end of another three weeks the soft palate and pharynx became again affected, the patient was unable to swallow, fluids regurgitated through the nose, and the paralysis spreading to the muscles of the limbs and trunk proved fatal at the end of another week. These cases, then, render it probable that the muscles of the back of the throat may be affected in the course of the primary disease, by the local inflammation, and at a later period by the form of paralysis, whatever may be its cause, to which the muscles of the body generally are liable. Of the remaining 125 cases, it is stated that in 16 cases the paralysis began soon, or a few days after the cessation of the sore throat, in 31 cases that it began in from 2 to 7 days, in 43 between 8 and 14 days, in 20 between 14 and 21 days, in 4 between 22 and 28, in one at the end of 40, and in another at the end of 60 days. It may, therefore, be stated broadly that in the majority of cases the paralysis begins in from 8 days to 3 weeks after every trace of local inflammation has subsided, while exceptionally it may be delayed as late as 8 or 9 weeks after the sore throat. The paralytic phenomena are said to be sometimes ushered in by general symptoms. Lorain and Lépine state that there is in some cases a sensible rise of the temperature of the body with acceleration of the pulse, but Maingault asserts positively that the temperature of the body remains at, or may even fall below the normal, and Weber insists upon a remarkable slowing of the pulse as a premonitory symptom. The truth appears to be that none of these phenomena are constant, and that the nervous symptoms often declare themselves without any warning. All authors are agreed that in by far the majority of cases the nervous disorders begin by paralysis of the veil of the palate, which declares itself by a nasal tone of voice, by fluids being ejected through the nose during attempts at deglutition, and by inability to pronounce clearly the explosive consonants. The patient can swallow solids better than fluids, inasmuch as the latter are more readily ejected than the former through the nose, but when the pharyngeal muscles become paralysed, as they often do, the difficulty of deglutition becomes greater and the patient is in danger of being starved from inability to swallow sufficient food, or of being suffocated by the bolus becoming impacted in the pharynx, the latter accident being facilitated by the fact that anæsthesia of the mucous membrane of the superior part of the larynx is often superadded to the pharyngeal and palatal paralysis. Of the 171 cases collected, disorders of the function of the palate were present in 128 and absent in 42 cases. Of the 128 cases in which such disorders were present a nasal quality of speech is alone mentioned in 6 cases, regurgitation of fluids through the nose alone in 5 cases, and difficulty in swallowing in 10 cases, leaving 109 cases in which a combination of two and generally of all the three symptoms was present. In none of these cases is it mentioned definitely that there was a difficulty in swallowing solids as well as fluids. Paralysis of the palate was accompanied in 30 cases by symptoms indicative of disorders of the muscles of the pharynx, tongue, or larynx. Paralysis of the abductors of the vocal cords gives rise to difficulties of respiration, and of the muscles which approximate and render tense the vocal cords, to alterations of voice, which may vary from a slight hoarseness to complete aphonia. The laryngeal disorder was declared in nine cases by paroxysms of coughing on swallowing, which was, therefore, probably caused rather by anæsthesia of the larynx than by paralysis of the vocal cords. The operation of tracheotomy is only mentioned as having been performed in two of these cases. It was declared in 5 cases by aphonia alone, and in three other cases the aphonia is said to have been accompanied by paralysis of the vocal cords, while in another three cases the vocal cords are stated to have been paralysed, but without mention being made of any disorder of voice. In 1 case the voice is said to have been toneless. In 10 cases it is stated that the patient had suffered from difficulties of articulation or that his speech was indistinct or embarrassed, but without giving details which would enable us to determine the particular group of muscles affected. The larynx or pharynx is said to have been affected in 6 cases, in which no mention is made of nasal speech or the other symptoms which indicate paralysis of the palate. In 1 case the abductors, and, two days later, the adductors of the left vocal cord were completely paralysed, and in a second case the adductors were paralysed so that there was complete aphonia, while in a third case incomplete closure of the glottis was declared by a cough which is said to have been non-explosive. Paralysis of the tongue is declared, when the affection is unilateral, by deviation of the organ on protrusion, and when bilateral by the inability of the patient to project the tip beyond the lips, or, in lesser degrees, by tremulousness on protrusion, and by a difficulty being experienced in executing the first act of deglutition. Unilateral paralysis of the tongue was present in two cases, as declared by deviation on protrusion, and in three other cases the tongue is said to have been feeble or paralysed, while in 3 of the cases reported by Boissarie the pharynx and tongue are said to have been paralysed, but the disorder of function by which the paralysis was declared is not described. Disorder of the seventh nerve was declared in 2 cases by twitchings or involuntary spasms of the facial muscles, and in 13 cases by more or less of paralysis. In 4 of these cases the paralysis affected the one side of the face only; in five cases the paralysis was bilateral, and declared chiefly by an obliteration of the lines of the face and an immobility of the countenance which gave to the patient an idiotic expression. In the remaining 4 cases the lips alone are mentioned as having been affected, and paralysis of these was declared by the inability of the patient to puff out his cheeks, to smack, or to blow out a candle. In one of these cases the paralysis was so great that, in addition to these symptoms, the lower lip was pendulous, and the saliva dribbled from the mouth. It is probable that in this case the masticatory muscles were affected along with the lips, and in one other case it is mentioned that the patient experienced great fatigue on attempting to chew his food.

The muscles of the eyes are those which are most usually attacked after those of the throat, and one or other of these have been affected in 77 out of the 171 of my collected cases. The disorder most frequently met with is loss of the power of accommodation for near objects, caused by paralysis of the ciliary muscles, which has been present in no less than 54 out of the 77 cases in which disorders of the ocular motor functions have been observed. This defect is declared by inability to read small print, but the disability is corrected by the use of convex glasses. In one case it is stated that the loss of accommodation was complete for two, and in another for six days, while in a third case it is stated to have lasted six days, in a fourth thirteen days, in a fifth a month, and in a sixth seven weeks. In the remaining 48 of the 54 cases there is no definite mention of the duration of this symptom. Disorders of the pupils are mentioned in 13 of my cases. In 2 cases the pupils are said to have been unequal, in 5 cases they were dilated, and in two of them the reaction to light was sluggish; in 4 cases they are stated to have been contracted, and in one of them the reaction to light was sluggish; in one case the reaction to light was sluggish, but no mention is made of the size of the pupil; and in another case it is stated that the circular muscle of the iris was completely paralysed on both sides, so that the pupils must have been dilated and reactionless to light and accommodation. Disorders of the movements of the eyeballs and eyelids were present in 30 cases. In 16 of these cases, the disorder was declared by double images, and in 10 cases by the presence of strabismus. In 4 of the last cases it is simply stated that the patient squinted, or was the subject of a strabismus; in 5 cases the strabismus was convergent, and in one case only divergent. In 2 cases the disorder consisted of double ptosis, without mention being made of any other motor visual disturbance; but in 4 cases a double ptosis, and in one case a unilateral ptosis is said to have been associated with diplopia. In one case of convergent strabismus, the eyeballs are said to have been in the position of exophthalmos, in another case exophthalmoplegia externa was present, and in a final case the disorder is described as a horizontal nystagmus.

The lower extremities were affected in 113 of my 171 collected cases. In 36 of these cases the lower extremities were either completely paralysed, or their motor power was so much diminished that the patient was unable to stand or walk, while in 50 cases the condition is described as one of weakness, feebleness, or great feebleness of the lower extremities, or is declared by distortion of the toes with or without other indications of paralysis. In the remaining 27 cases the condition of the limbs was declared chiefly by a disorder of gait. In 18 of these cases the disorder is described as a vacillating, uncertain, or staggering gait, and in 9 cases the gait is said to have been ataxic. In 4 of these last cases the ataxic gait is stated to have been accompanied by weakness of the extremities, and in none of them is it mentioned that the muscles were free from all traces of paralysis, and until we meet either in our own observations or the observations of others with more cogent evidence than any hitherto adduced we refuse to believe in the occurrence of genuine ataxia as a sequel to diphtheria. In 2 of the cases the paralysis was limited to one of the lower extremities, and in other 2 cases the muscular feebleness was accompanied by twitchings of the legs. In one case the affected extremity is said to have been the subject of chorea-like movements, and in another case the limbs are said to have been affected by trembling like those of a drunken man. In seven cases swaying movements and inability to stand with closed eyes is mentioned.

The upper extremities were affected in 60 out of the 171 cases. In 23 of these cases the paralysis was either complete, so that the limbs fell when moved like inert masses, or so great in degree that the patient was not able to raise his arms or to use his hands in feeding himself. In the remaining 37 cases the condition of the upper extremities is described as a feebleness of the arms or of the grasp, or as a weakness or helplessness of the hands. In two of these cases the deltoids were affected the most, and in one of these the muscles are said to have been shrunken. In another case the shoulders are said to have been contracted, a condition which would most probably result, not from active muscular spasm, but from a predominance of the action of more or less healthy adductors over that of paralysed deltoids. In one case the muscular feebleness was accompanied by twitchings of the limbs, in another case there was stiffness and twitching of the left arm, and in two cases the hands are said to have been the subject of tremors like chorea.

The muscles of the trunk, chest, or neck were affected in 58 out of the 171 cases. In 19 of these cases it is expressly stated that the muscles of the trunk were affected; in 10 of these cases the muscles are said to have been paralysed, that the paralysis was general, or that the patient was unable to sit; in 5 cases it is said that the muscles of the trunk were feeble, or that the body was weak; in 2 cases the paralysis was declared by the presence of saddle back when the patient stood erect, and in 1 case by a stooping forward of the body and head when sitting, while in a final case it is mentioned that the lumbo-sacral muscles were paralysed. The muscles of the neck were affected in 15 cases, the paralysis being declared by flexion or drooping of the head when the patient was sitting, or by a complete inability to move the head. Implication of the muscles of the neck is mentioned alone in 9 cases, in association with paralysis of the muscles of the trunk in 2 cases, and with the muscles of respiration in 4 cases. The muscles of respiration are expressly mentioned to have been affected in 33 cases; in 14 of these the patients are said to have suffered from embarrassment of breathing, asthmatic attacks, dyspnœa, or to have died from asphyxia. In 1 case the respirations are said to have been slow, in a second the respirations were 24, in a third 40, and in a fourth 50 in the minute. In 1 case the respirations are said to have been excessive, in a second superficial, in a third the breathing was intermittent, in a fourth it was Cheyne-Stokes in character. The diaphragm is stated to have been paralysed in 4 cases, while the breathing was thoracic in 3 cases. In 2 cases the intercostal muscles are stated to have been paralysed, and in one of these cases it is mentioned that the breathing was abdominal. In 2 cases it is stated that the muscles of expiration were affected, and the authors give their adhesion to Duchenne's theory that in these cases it is the intrinsic muscles of the lungs which are paralysed; but it does not appear to me an improbable view that the respiratory difficulties were caused in them by a complicating bronchitis. In 25 of the 33 cases in which the muscles of respiration were involved in the paralysis the disease proved fatal.

Sensory disorder very generally accompanies the loss of motor power in diphtheritic paralysis. In 86 of the 171 cases here collected no mention is made in the records of the presence of sensory disorder; in 49 of the cases the patients were children under 10 years of age, so that the presence of numbness or a slight degree of tingling in the fingers and toes would be readily overlooked; in 13 cases in which the patients were 10 years and upwards the disease proved fatal in a few days, so that any sensory disorder which might have been present would be overshadowed by other important symptoms; in 4 cases the motor affection consisted of loss of visual accommodation only, and in most of the remaining 18 cases the paralysis either did not extend to the limbs, or the record is manifestly imperfect. It may, therefore, be concluded that the motor paralysis is almost always, if not always, accompanied by some disorder of sensibility. It is, indeed, asserted by Sanné that paralysis of the soft palate and of the muscles of the pharynx and larynx is usually preceded by some degree of anæsthesia of the upper part of the larynx, which is indicated by the presence of a short abrupt cough, caused by the passage of particles of food through the glottis, while the motor disorders of the extremities are also often preceded by numbness and other paræsthesia in them. In 56 out of the 85 cases in which sensation was affected the disorder consisted of numbness, formication, tingling or creeping sensations, which usually began in the toes and fingers and extended upwards over the feet and hands as far as the knees and elbows, and, in occasional cases, as far as the hips and shoulders. In one of these cases the patient complained of a disagreeable sensation of tingling in the tip of the tongue; in a second the cheeks and nose felt numb; in a third the patient experienced pricking sensations in the tongue, which felt as if it were swollen; in a fourth there was numbness of the tip of the nose, glans penis, scrotum, and anus; in a fifth the upper lip felt numb; and in a sixth the patient complained of numbness of the upper lip, back of the tongue, and back of the neck. In one case numbness of the lower extremities was accompanied by a sensation as if the feet were surrounded by bandages, and in another case the patient felt, particularly after washing, as if grains of sand were lodged in the tips of the fingers and under the nails. In every case in which the patient complains of a considerable degree of numbness there is doubtless some impairment of the tactile sensibility in the affected part, but disorder of this form of sensation is only specially mentioned in 14 cases; in 10 of these cases the tactile sensibility is said to have been diminished, impaired, or lost, chiefly in the hands and fingers, or it is stated that the patient could not feel or distinguish objects, while in 2 cases it was diminished in the tongue and lips, in 1 case it was lost in the tongue, lips, and anterior part of the palate, and in 1 case it was lost in the tongue, lips, and soles of the feet. The muscular sense was diminished or lost in 6 cases; in 2 of these cases it is stated that the sense of the position of the limbs on the eyes being closed was diminished or lost, in 1 case that the muscular sense was diminished, and in 2 cases that objects escaped from the hands on the eyes being closed. In 2 cases it is said that there was retardation of the conduction of sensations of heat and cold. The sense of pain is mentioned as having been affected in 27 cases, and in 24 of these one or more of the paræsthesiæ just described were also present. In 15 of these 27 cases various painful sensations were complained of, these being situated chiefly in the limbs, trunk, neck, and around the chest. In 2 of these cases, however, the pain is said to have been evoked on the slightest touch or the slightest movement, while in 3 cases it is stated that the patient suffered from tearing pains in the joints, pains in the knee, or rheumatic pains in the limbs. The electro-muscular sensibility is mentioned in 4 cases; in 3 cases it is said to have been lost generally, or in the calves; and in 1 case the application of the galvanic current caused great pain in the extremities. The nerve trunks are said to have been painful to pressure in 3 cases; in 1 of these the superior ganglion of the sympathetic cord in the neck is said to have been also tender on pressure. The sense of pain was impaired or lost in 6 cases; in 5 of these the analgesia is said to have been complete, but in the remaining one it was slight in degree. The general cutaneous sensibility was affected in 41 cases; in 4 of these the disorder was one of hyperæsthesia, which appeared either in the form of an exaltation of the cutaneous sensibility of the body generally, or was limited to the hands, to patches on the hands and feet, or to one side of the body. The cutaneous sensibility was diminished or lost in the remaining 37 cases; in 13 of these cases it is mentioned generally that the cutaneous sensibility was diminished or lost, or that there was partial or complete anæsthesia, without the parts affected being specified; in one case it is stated that the surface of the arms, legs, back, and breasts was anæsthetic; in 12 cases the anæsthesia was limited to the extremities, often to the hands and feet, sometimes extending to the knees and elbows, or embracing the whole limbs, and in one case involving the cheeks and nose as well as the extremities. In 1 case this anæsthesia was limited to the soles of the feet, and in another case it is mentioned that the patient could not feel the ground under him, but in this case it is probable that the muscular sense would be abolished as well as the cutaneous sensibility.

In 5 cases the anæsthesia was limited to the veil of the palate, in 1 case to the upper part of the larynx, and in another to the fauces, while in another the pharynx and back part of the tongue were anæsthetic as well as the soft palate. In 2 cases the anæsthesia was restricted to one side of the body, and it is probable that the condition was one of hysterical hemi-anæsthesia. The record of one of these cases is particularly suggestive of hysteria. A woman, aged 21 years, aborted after an attack of diphtheria, and two days later she was suddenly seized with right-sided hemiplegia and hemi-anæsthesia, which was cured in an hour by cutaneous faradisation, and although the patient died some days later from paralysis of the respiratory muscles, there can be little doubt that the transient hemiplegia and hemi-anæsthesia was a functional disorder.

The special senses are occasionally affected. In addition to the disorders of vision, already described, from paralysis of the ciliary muscles, and of the ocular motor nerves, it is stated in 3 cases that amaurosis was present for some days, and it is probable that in those cases the loss of vision would have been caused by changes in the optic nerves and retinæ. Bouchut* states that in such cases he has observed anæmia with serous infiltration of the retinæ. The same author also affirms that in exceptional cases of diphtheritic paralysis he has observed a true optic neuritis, with consecutive atrophy, but his statements have, so far we know, not yet been confirmed by other observers. Disorders of the sense of hearing is mentioned in 3 cases; in 1 case it consisted of tinnitus, in the second of tinnitus and deafness, and in the third of deafness alone. It is possible that these disorders may have been caused by catarrh of the Eustachian tube and middle ear, and are not, therefore, to be regarded as belonging to the post-diphtheritic nervous disorders. Taste was impaired in two cases, but disorder of the sense of smell is not mentioned as a symptom in my cases.

The *reflex actions* have not been carefully examined in a large number of my collected cases. The reflex contraction and associated movements of the palate are only mentioned in 36 out of the 128 cases in which paralysis of the soft palate was present. In 7 of these cases it is stated that the reflex action of the soft palate was diminished or that the palate contracted but little on reflex irritation, while in the remaining 29 cases it is stated that the reflex action of the palate was lost. In 16 cases of the last group it is simply stated that the reflex was lost, or that the palate was immovable on direct irritation; in 5 of the cases it was immovable to every form of excitation on one side, but some degree of contraction could be obtained on the other side either on direct irritation or on phonation; in 3 cases it is stated that the associated movements of the palate, such as the movement on deep inspiration and on phonation, were lost, as well as the power of contraction to direct irritation; in two cases tickling of the fauces caused nausea and retching, even although the reflex contraction to direct irritation was lost; and in 3 cases the palate contracted slightly on phonation, but not to direct irritation.

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* Bouchut (E.). "Atlas d'ophthalmoscopie médicale et de cérébroscopie," Paris, 1876, Figs. 105 to 108, p. 34. "Hemiplégie diphthéritique droite cérébroscopie; atrophie papillaire et embolie dans l'œil gauche; névrite congestive droite."—*Gaz. des Hôp.*, Tome XLII., 1869, p. 401; et "Signes ophthalmoscopiques dans les paralysies diphthéritiques."—*Gaz. des Hôp.*, Tome LVIII., 1875, pp. 667 to 673.

The condition of the cutaneous reflexes is only mentioned in 13 cases. In 4 of these it is stated that the reflexes were active, the cremasteric being maintained in one and the plantar in another, while in 2 cases it is stated generally that the superficial reflexes were present. In the remaining 9 cases, one or other of the cutaneous reflexes is said to have been diminished or lost; in 4 of these cases it is stated generally that the reflex actions were lost or almost lost, and in 3 other cases that they were feeble or diminished, while in one of these last cases the reaction to tickling was also diminished. In 1 case the plantar and epigastric reflexes are said to have been lost, and in another case the abdominal reflexes were absent.

The patellar tendon reactions were examined in 36 cases. In 28 of these cases these reactions were absent on both sides, and in two cases completely absent only on one side; in one case the reactions are said to have been almost completely lost; in three cases they were normal, and in two cases they were exaggerated.

The electrical reactions were only examined in 23 of the 171 cases. In six of these cases it is stated that the electrical (meaning most likely the faradic current) reactions were normal, diminished, or impaired, or that a strong current was required to obtain a minimum contraction, and in one case the electrical irritability of the digital nerves are said to have been lost. In seven cases the reactions to the faradic current are alone mentioned; in two of them the faradic contractility is said to have been intact or retained, in one case it was diminished in the arms and legs, in a second it was almost extinguished in the upper but present in the lower extremities, in a third it was diminished in the muscles of the hands, and in a fourth and fifth it is stated to have been lost in the soft palate. In two cases the galvanic excitability was alone examined. In one of these cases it is said that the galvanic irritability in the affected muscles was diminished, and in the other that the qualitative changes, which constitute the reaction of degeneration, were present. In seven cases the reactions to both the faradic and galvanic currents were examined. In one of these cases the faradic contractility is said to have been retained, but the galvanic contractility manifested the reaction of degeneration; in one case the faradic irritability was maintained and the galvanic lowered, but there was no reaction of degeneration. In four cases it is said that the faradic irritability was diminished, or that a strong current was required to evoke a minimum contraction, and that the galvanic irritability of the muscles showed the qualitative changes which indicate the reaction of degeneration; and in one case the irritability to both the faradic and galvanic currents is said to have been completely lost in the soft palate. From the above analysis of cases, it may be concluded that in the slighter degrees of paralysis the electrical reactions are either normal, or only slightly diminished in the nerves and muscles most affected; that in the more severe cases the faradic irritability of the nerves and muscles is lowered, while the galvanic irritability of the muscles is such that the anodal closing contraction is equal to, or exceeds, the cathodal closing contraction, this being the kind of formula named the partial reaction of degeneration. It is obtained when the affected nerves and muscles have undergone a degree of degeneration, which is not very profound, and from which recovery takes place in from six weeks to two months from the time at which the paralysis has ceased to progress. In the few cases in which the irritability to the faradic current or to both currents was lost, a high degree of degeneration is indicated, and in two of these cases recovery was delayed till the end of five months in the one, and six months in the other. In the remaining cases, however, recovery took place in from 40 days to four months, but in these cases the condition of the reactions in such small muscles as those of the soft palate and of the hands is alone mentioned, and it is possible that some degree of paralysis might have remained in them even after the patient is said to have recovered.

The *trophic* changes consist chiefly of wasting of the paralysed muscles, but this is mentioned only in 12 of the 171 cases here collected. In 2 of these cases it is said that there was great pallor and wasting, so that the condition might have been one of general emaciation; but in the remaining 10 cases it is expressly mentioned that the affected muscles were atrophied, or that there was a progressive wasting of the paralysed muscles. In one of the last group of cases atrophy was most pronounced in the small muscles of the hands; in a second the muscles of the calves of the legs were affected chiefly, while in a third case the tongue is said to have been wasted and the subject of fibrillary contractions. In one case the left knee and the joints of the left hand were swollen and tender. Extreme pallor or anæmia is said to have been present in 6 cases, and as this symptom does not find an explanation in these cases in the presence of an exhausting diarrhœa or other complication, it must be regarded as in some way resulting from the morbid process which underlies the paralysis. *Secretory* disorders are not often mentioned, but in one case perspiration is said to have been suppressed in the feet, and in another that the patient suffered from heavy perspirations. *Vaso-motor* phenomena are also not mentioned, except in a few cases, in which it is said that the extremities were very cold, or in which the hands and feet were of a livid colour. The temperature has only been raised above the normal in a few cases, and the elevation of temperature was due to a complication of bronchitis or pneumonia, or of some other acute disease.

The *visceral* functions are often profoundly affected in diphtheritic paralysis. We have already seen that the respiratory muscles are frequently involved, and that the disease often proves fatal by respiratory paralysis, and we shall now find that the heart is no less frequently attacked. Disorders of the circulation are mentioned in 43 of the 171 cases here collected, the cardiac disturbance being declared in 17 of these cases by a feeble, irregular, and frequent pulse, in 15 cases by feeble and slow pulse, and in 11 cases by some disorder in the functions of the heart. Of the 17 cases in the first group the increaaed frequency of the pulse was accompanied in five cases by elevation of temperature and some acute disease, such as pneumonia, tuberculosis, and collapse of the lung, so that the alteration in the rate and strength of the pulse might possibly be caused by the intercurrent disease, but the remaining 12 cases were free from all complications, and consequently the disorder of the circulation in them must be regarded as being caused by implication of the nerves of the heart in the paralysis. Of these 12 cases, it is stated in one that the pulse, which was 80 when the patient was lying down, rose suddenly to 135 when she sat up in bed; in a second that the pulse rose from 80 to 100 on the slightest exertion, and in a third it is simply said that the pulse was frequent. These patients recovered. In the other 9 cases it is stated that the pulse varied in frequency from 80 beats in the minute, the lowest to 164 beats—the highest counted number of beats—although the use of the terms, extraordinary rapidity and great frequency with irregularity, would seem to indicate that the pulse had sometimes attained to a higher rate of frequency than is expressed in numbers. In addition to its frequency, the pulse is said to have been irregular, feeble, small, and often imperceptible, and all of these patients died from collapse or gradual cessation of the heart's action. In one of these cases the cardiac disturbance was accompanied by dyspnœa, and in another by Cheyne Stokes' breathing. Of the 15 cases in which the pulse was feeble, irregular, and slow or infrequent, the beats varied in 11 cases from 80 to 42, and to 14 beats in the minute in one case, and the patients subsequently recovered. In 5 cases the pulse beat from 70 to 14 in the minute, and the patients died from cardiac failure; and in one of these cases cyanosis was present near the fatal issue, and the extremities were cold. Of the 11 cases in which functional disturbances of the circulation are mentioned, in 2 cases a systolic murmur was present, in 1 case the patient suffered from palpitation, and in another case the patient suffered from pain in the præcordial region which extended from the sternum to the left shoulder and also down the left arm, but in all recovery took place. In 5 cases the patients died suddenly from fainting attacks or syncope; in 1 case the patient is said to have sunk suddenly, and in another fatal case the patient suffered from dyspnœa and organic disease of the heart. In many of the cases in which the death was caused by failure of the circulation, the cardiac disorder, whether declared by a feeble, irregular, and frequent, or by a feeble and infrequent pulse, was accompanied by severe vomiting and severe gastro-intestinal pain, either of the nature of gastralgia or of colic. In 22 cases disorders of the functions of the bowels or bladder are mentioned. In twelve of these cases the symptoms consisted of constipation alone; in one of these cases it is said that the bowels were obstinately constipated for eight, and in another case for as long as twenty-four days. In the remaining ten cases, the urine was passed involuntarily in two cases, the bladder was imperfectly emptied in two cases, the bladder and rectum are said to have been paralysed in four cases, and the rectum alone in two cases. It is mentioned in six cases that complete impotence was present, and in one of these cases it is said that this function was in abeyance for many months.

Course, Duration, and Termination.—The course of diphtheritic paralysis is somewhat variable. The sensory disorders are apt to undergo great variations, both in intensity and extent, in the course of the disease, and although the motor disorders only undergo gradual changes of increase or decrease, yet different groups of muscles become successively invaded, so that as the paralysis is disappearing in one set of muscles, others are being attacked. In 14 out of the 171 cases here collected, the paralysis was limited to the muscles of accommodation, to the external ocular muscles, or to the vagus, as indicated by cardiac disorders, and neither the soft palate nor the muscles of the extremities were affected, and in 29 cases the soft palate or the muscles of the pharynx and larynx were alone affected, while the extremities were free. In 106 cases the muscles of the palate, pharynx, or larynx, and those of some or of all the limbs were invaded; in 35 of these cases the muscles of the throat were attacked first, those of the lower extremities second, and those of the upper extremities last; in 33 of the cases the muscles of the throat were attacked first, the lower extremities second, while the upper extremities are not mentioned as having been affected; in 10 cases the muscles of the throat were first affected, while later those of the lower and upper extremities were simultaneously attacked; in 2 cases the muscles of the upper extremities were invaded after those of the throat, and those of the lower extremities were the last to be attacked; in 4 cases the muscles of the legs were invaded after those of the throat, and the arms and hands remained free; in 6 cases the muscles of the legs remained free, while those of the throat and upper extremities were successively invaded; and in 2 cases the muscles of the throat, legs, and arms were simultaneously attacked. In 3 cases the extremities were simultaneously attacked at the onset, and the muscles of the throat were the last to be affected; in 2 cases the lower extremities were the first to be paralysed, the throat second, and the upper extremities last; in 3 cases the lower extremities were attacked first, the upper second, and the throat last; in 1 case the upper extremities were attacked first, the lower second, and the throat last; in 3 cases the muscles of the lower extremities were first attacked, the throat second, while the upper extremities remained free; and in 1 case the muscles of the eyeballs were attacked first, those of the throat second, those of the upper extremities third, and those of the lower extremities last. In 22 cases the muscles of one or more of the limbs were attacked, while those of the throat remained free; in 8 of these cases the paralysis began in the lower extremities, and the upper were subsequently invaded; in 5 cases the lower and upper extremities were simultaneously attacked, and in 9 cases the lower extremities were alone affected.

The duration of the disease is also very variable. Of my 171 cases 126 recovered and 45 died. Of the 126 cases which recovered the duration of the paralysis is not mentioned in 16 cases. In 8 cases it is measured in days, in 14 cases in weeks, and in 88 cases in months, dating from the commencement of the sore throat. Of the 8 cases in which the paralysis lasted only a certain number of days, in 2 cases its duration is said to have been a few days only, and in 6 cases 3, 13, 14, 24, 33, and 40 days respectively. In these cases the paralysis was restricted to the muscles of the throat and those of the eyes, while the limbs were free. Of the 14 cases in which the duration of the paralysis is measured in weeks, in one case it is said to have lasted some weeks, in 6 cases from 3 to 6 weeks, in 4 from 7 to 10 weeks, and in 3 cases it lasted 12, 13, and 15 weeks respectively, the average duration of the 14 cases being 8 weeks. Of the 88 cases in which the duration of the paralysis is measured by months, in 1 case it is said to have lasted some months, in 16 cases from 1 to 1½, in 19 from 2 to 2½, in 10 from 3 to 3½, in 20 from 4 to 4½, in 15 from 5 to 5½ months, in 4 cases 6 months, and in 4 cases 7, 8, 9, and 12 months respectively, the average duration of all the cases being 3½ months. In estimating the duration of the disease, however, it must be remembered that a large number of the cases disappear from view before recovery is complete, and if all the cases in which the paralysis had become general had been closely observed until every trace of paralysis had disappeared, it is probable the average duration would be at least 4 months from the commencement of the sore throat. In the list of cases from which the above analysis was made, the longest duration of the paralysis is 12 months; but Maingault mentions a case of generalised paralysis, not included in this list, in which it lasted 20 months, and in a case already reported by myself, also not included in the list, the patient was unable to use his hands in agricultural work 18 months after he had suffered from an attack of sore throat. As a rule, the paralysis of the veil of the palate disappears in from 10 to 15 days, but occasionally it persists for some months, and Morisseau[*] records the case of a girl who had suffered at 8 years of age from an attack of diphtheria and subsequent paralysis, and whose speech had a slightly nasal quality at 17 years of age. Turning now to the 45 cases in which the disease proved fatal. In 2 of these cases that died 8 and 13 days respectively from the commencement of the paralysis the cause of death is not stated. In 8 cases death was caused by an intercurrent disease, such as lobar or lobular pneumonia, tubercle of the lung, albuminuria and anasarca with œdema of the lungs and empyema, in eight cases death occurred suddenly from syncope, in 10 cases from cardiac failure or asthma, in 14 cases from respiratory paralysis, and in two cases from asphyxia caused by food finding its way through the glottis, the bolus lodging in the trachea in the one, and in the left bronchus in the other case.

Morbid Anatomy.—An autopsy was obtained in 19 of the 45 fatal cases. In three cases the results of the autopsy were negative, in six cases the examination simply showed that the patient died from the impaction of a bolus of food in the air passages or some intercurrent disease like pneumonia, tubercle of the lungs, or œdema of the lungs with congestion of the kidneys, while in 10 cases changes were discovered in the peripheral nerves, the nerve roots, or the anterior grey horns of the spinal cord. In a fatal case of diphtheritic paralysis examined by Charcot and Vulpian[†] in 1862, the authors found a degeneration of the nerves of the palate which was characterised by the medullary sheath being broken up into globular masses and granules, just as occurs in the peripheral portion of a nerve after section. These observations were some years later confirmed by Lorain and Lépine,[‡] and Liouville[§] found similar changes in the phrenic nerve in a case that died from paralysis of the diaphragm. In 1871 Oertel,[‖] in a case of general muscular atrophy after diphtheria, found diffused hæmorrhages in the spinal membranes and in the grey substance of the spinal cord with moderate nuclear proliferation of the neuroglia. The vessels and nerve fibres of the anterior and posterior nerve roots were surrounded by fatty granules. Micrococci were present in the blood and tissues. In 1876 Pierret[**]

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[*] Morisseau. "Paralysie du voile du palais comme cause du nasonnement."—*L'Union Médicale*, October, 1851, No. 126.

[†] Charcot et Vulpian. *Comptes rendues de la Soc. de Biol.*, 1862.

[‡] Lorain et Lépine. *Op. cit.*, p. 608.

[§] Liouville (H.) *Bulletin de la Société Anatomique*, 1869-70.

[‖] Oertel. *Loc. cit.*, 248.

[**] Pierret. *Société de Biologie*, 23 Déc., 1876.

found the spinal membranes covered by a pseudo-membranous exudation which compressed the nerve roots and caused a consecutive ischæmia of the spinal cord by obliteration of the vessels of the pia mater.

Soon afterwards Vulpian,[*] while failing to confirm Pierret's observations, found a rarefaction of the connective tissue of the posterior and external portion of the anterior horns of the spinal cord, while the ganglion cells had become globular and homogeneous, and their nuclei showed signs of multiplication. In a case of diphtheritic paralysis, reported as one of acute ascending paralysis, Westphal[†] found the cerebral pia mater of a milky appearance, but the medulla and spinal cord were normal. The anterior crural nerves manifested on microscopical examination changes similar to those found in the musculo-spiral nerve in a case of lead paralysis. The nervous tissues were carefully examined by Déjèrine in 1877-78 in several fatal cases of diphtheritic paralysis. He found in five cases degenerative neuritis of the anterior roots of the nerves on a level with the portion of the cord from which the paralysed muscles were innervated; in two of these the posterior roots on the same level were similarly affected. In three of the cases the ganglion cells of the anterior grey horns showed slight changes, and in one case the anterior horns themselves are said to have been slightly altered, while no mention is made of the cells or of the grey matter in the fifth case. In one case evidences of neuritis were found in the sciatic nerves. Meyer[‡] found evidences of a parenchymatous neuritis of the peripheral nerves and nerve roots, and in the bundles of nerve fibres which pass through the spinal ganglia. He also observed that many of the ganglion cells of the anterior horns had lost their processes and become rounded, while they contained fatty granules. Similar changes were found by Dr. Abercrombie[§] and Dr. Kidd[||] in the ganglion cells. In a case reported by Kast[**] the hypoglossal and spinal accessory nerves in one by Krauss[††] the oculo-motor, and in one by Suss[‡‡] the pneumogastric nerves were degenerated, while Henoch met with degeneration of the nerves and muscles of the heart.

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[*] Vulpian (A.). "Maladies du système nerveux."—Rec. par Bourceret. Paris, 1879, p. 167.

[†] Westphal (C.). "Ueber einige Fälle von acuter tödtlicher Spinallähmung sogenamiter acute aufsteigender Paralysæ."—*Archiv für Psychiatrie*, Bd. VI. Berlin, 1876, p. 765.

[‡] Meyer (P.). "Anatomische Untersuchungen über diphtheritische Lähmung."—*Virchow's Archiv*, Bd. LXXXV., 1881, p. 198, et seq.

[§] Abercrombie (John). "On diphtheritic paralysis in children."—*Transactions of the International Medical Congress*, Vol. IV., Loud. 1881, p. 63.

[||] Kidd (Percy). "A contribution to the pathology of diphtheritic paralysis."—*The British Medical Journal*, Vol. I., 1883, p. 57.

[**] Kast. *Loc. cit.*, p. 47.

[††] Krauss. *Loc. cit.*, p. 490.

[‡‡] Suss. *Op. cit.*, p. 25.

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(*b*) PARALYSIS AFTER TYPHOID FEVER.

THE occurrence of paralysis during convalescence from the continued fevers was known to Hoffmann, Cullen, Sauvages, and many of the older authors, but it was not until the time of Landry and Gubler that the doctrine of post-febrile paralysis had assumed a definite form. The paralysis is at times manifestly caused by cerebral disease, most commonly a thrombosis of an artery or of a vein, and at other times by disease of the spinal membranes or of the spinal cord itself; but after eliminating these varieties of paralysis we have been able to collect from the subjoined sources a large number of cases in which the symptoms have most probably resulted from disease of the peripheral nerves. The records of many of these cases, however, are so imperfect that it would be a fruitless task to enter upon a detailed analysis of them. The conviction, has, indeed, been forced upon our mind that the material for the induction of positive conclusions with regard to the relation between typhoid fever and the continued fevers generally and multiple neuritis does not at present exist, and that the doctrine can only be placed on a secure basis by new and carefully recorded observations. As a starting point for future investigations, we propose to give a brief review of the more important published observations, to mention two cases which have come under our own observation, and finally to state such conclusions as the recorded facts appear to warrant.

Gubler has reported several cases of local paralysis consecutive to typhoid fever. In a boy, aged sixteen, a few days after the cessation of the fever, it was noticed that he spoke with a nasal twang, and this was found to depend on paresis of the palate. A little later the patient could not see to read clearly, and he was obliged to hold a book further away than usual in order to distinguish the letters. This is the only example known to me of paralysis of accommodation following typhoid, and although the details of the case are somewhat scanty, the recorded fact may be remembered in connection with a remark of Dr. Gowers, that paralysis of accommodation never occurs as a result of typhoid fever.

Another boy of the same age, after an attack lasting forty-seven days, complained of pains in the legs, and a fortnight later he was unable to raise himself in bed; the lower limbs were feeble, trembled, exhibited fibrillary contractions, and their muscular irritability was greatly increased. The hands were also weak, and sometimes the difficulty in grasping was augmented by marked stiffness of the fingers. The speech resembled that of general paralysis.

Meyer reports the case of a boy who, during convalescence from typhoid, noticed weakness of the whole right side of the body; it felt cold and numb, and there were pains in the shoulder and upper arm during changes in the weather. The limb became weak and wasted. When seen by Meyer, about 10 months after the onset of the fever, there was tremor of the right hand during voluntary movements, wasting of the deltoid and marked atrophy of the right forearm and hand, especially of the interrosseous muscles and the thenar and hypothenar eminences. The hand had a claw-like shape, the fingers could not be abducted, adducted, or fully extended. The electric sensibility of the whole of the right side, including the right side of the tongue, was diminished; the contraction of the muscles to electricity was less active than normal, and this was particularly noticeable in the deltoid, the extensor indicis, and the small muscles of the hand. After eight months' treatment with the faradic current the limb regained power almost completely, but there was still some impairment of the electro-cutaneous sensibility, and the muscles on the right side were more wasted than on the left side. In this case, while the right hemianæsthesia was apparently of central origin (possibly a hysterical phenomenon) the atrophic paralysis of the deltoid and other muscles is, perhaps, better explained by disease of some of the peripheral nerves than of their roots or nuclei of origin.

Surmay mentions two cases of local paralysis after typhoid. In one case there was feebleness of the extensor muscles of the hands and fingers, together with complete paralysis of the extensors of the toes. In the other case weakness of the right leg was followed by weakness and subsequently complete paralysis of the left leg.

Handfield Jones published the case of a child 12 years old, in whom paresis of the lower limbs developed after a severe attack of typhoid. The paresis, more marked in one leg than in the other, was associated with some hyperæsthesia, with disturbances of the temperature sense, and with contractures of the gastrocnemii. The electrical contractility of the muscles of the lower limbs was almost abolished. These symptoms gradually disappeared in two and a half months, the sensory disorders going before the weakness, and the contractures last of all.

Kraft-Ebing mentions weakness of the adductors of the thigh, with hyperalgesia of parts supplied by the saphenous nerve.

Bailly records a few cases of loss of power in the lower limbs, the weakness being sometimes associated with anæsthesia, and with contractures. In one of his cases atrophy of the shoulder muscles is mentioned; in two other cases paralysis of the veil of the palate.

Nothnagel observed during the war of 1870-71 several cases of paralysis after typhoid. In four cases the territory of one of the ulnar nerves was affected; the ulnar side of the hand had lost its sensibility, and the muscles supplied by the nerve were feeble and atrophied and gave diminished reaction to both the faradic and galvanic currents. In two of these cases no mention is made of other nerve symptoms besides the ulnar paralysis, but in the remaining two there were other noteworthy nerve symptoms. In one of these cases, in addition to the ulnar paralysis, the patient complained of radiating pains in the shoulder of the affected side, and of pains in both the lower extremities, accompanied by a feeling of fatigue in walking; and in two other cases the patient suffered from pains in the calves of the legs and loss of feeling in the feet, symptoms which after a time were supplanted by a painful feeling in the external surface of the right thigh, but without any motor disturbance. In a fifth case Nothnagel found anæsthesia and weakness of the left arm, the weakness being most pronounced in the muscles of the shoulder; there was complete recovery in eight months. He also reports four cases in which the lower limbs were partially paralysed, the feebleness being associated with partial anæsthesia and neuralgic pains, together with creeping and tingling sensations. In one of these cases paresis and tremor of the lower limbs was followed by weakness of the upper limbs; in another case the patient, at the beginning of convalescence, experienced a feeling of numbness and creeping in the left leg, and subsequently paralysis of the limb gradually developed; then the extensors of the right hand became paralysed, and four days later some of the muscles of the left hand. Sensibility was diminished in the affected extremities.

Murchison says: "Paralysis is an occasional sequel of enteric fever. It may not supervene till many weeks after the commencement of convalescence, and it is usually temporary, recovery taking place within a few weeks or months. Now and then these attacks of paralysis, particularly in the legs, terminated in atrophy of certain of the muscles, and I have met with several instances where permanent distortion has resulted. One of my patients, a girl aged 18, had all the signs of paralysis of the right third nerve in a marked degree throughout an attack of enteric fever. This had first occurred fourteen years before, after an attack of measles, but for many years had almost disappeared until the seizure with enteric fever, on convalescence from which only slight ptosis remained."

Leyden records a case in which marked atrophy and paralysis of the muscles of the right arm and shoulder followed typhoid, the motor symptoms being ushered in and accompanied by very acute pains. The electric contractility of the muscles was much diminished. After two months' treatment with electricity the limb recovered almost completely. He also refers to a case published by Bénédict, in which there was paresis of the right lower limb, with atrophy of the quadriceps femoris.

Eisenlohr. A man, aged 30, about eleven days after the temperature became normal, had weakness and numbness in the left leg and foot, together with violent pains in these parts, and in both knees. The next day there was effusion into the right knee, and the temperature rose to 104° F. There was also diminished sensation, and feebleness of the muscles in the territory supplied by the left peroneal nerve. On the 14th day the left knee was swollen, on the 16th day the right elbow became painful and swollen, whilst the swelling of the left knee subsided. The muscles supplied by the left peroneal nerve showed diminished reaction to the faradic current. The left foot was œdematous, and in the position of equino varus.

On the 24th day the flexors of the foot and the extensors of the toes were completely paralysed, and gave the reaction of degeneration to electricity.

Eisenlohr thinks it probable that the paralysis was due to impaired function of the branches of the left sciatic nerve, in consequence of effusion into their sheaths, but he does not discuss the nature of the joint affection. Some writers quote the case as an example of paralysis coming on during a relapse of typhoid fever. But the association of a migratory joint affection with pyrexia suggests an attack of acute rheumatism. It seems probable that the joint affection and the paralysis, coming on almost simultaneously, were due to the same cause, but whether that cause was the organism or poison of typhoid fever, or the agent which sets up rheumatism, must we think remain an open question.

Bernhardt has published the case of a young man who, during convalescence from typhoid, suffered from pains along the ulnar border of both forearms; these were followed by an atrophic paralysis of all the muscles supplied by both ulnar nerves, so that the interosseous spaces, the thenar and hypothenar eminences became greatly wasted, and the reaction of degeneration was obtained in the affected muscles. Anæsthesia too was present in the ulnar territory. Complete recovery ensued after three months' treatment with electricity.

Baümler, in his able paper on paralysis of the serratus magnus, gives full details of the case of a man, aged 50, who two months after the onset of convalescence from typhoid, suffered from burning pains in the neck, quickly followed by weakness in the right arm, and subsequently of the left arm. When examined three months later, complete paralysis of the *right* serratus magnus was found, together with an atrophic paralysis of the muscles supplied by the *left* muscular spiral nerve, with the exception of the supinator longus. There was slight analgesia and paræsthesia of the left thumb and index finger, and somewhat later paralysis and atrophy of the anterior part of the left deltoid. Spontaneous recovery set in ten months after the first paralytic phenomena had been observed, and in another six months the muscles had completely recovered their bulk and power. The sensory disturbance in the left thumb, however, persisted, though to a less marked degree than formerly. Baümler believes that the symptoms were due to a spinal lesion, but the presence of sensory disturbances and the complete recovery speak much more in favour of a peripheral neuritis, mainly affecting the right long thoracic and the left muscular spiral nerves.

Mitchell gives a brief record of a case of paraplegia associated with muscular atrophy and contraction, which occurred during convalescence from a protracted attack of typhoid. After a two months' course of massage and the constant current, the lower limbs regained power to a considerable extent. The case was complicated by the presence of tremor, which Mitchell considered hysterical in nature, but he says the paralysis points to some degeneration of the cells of the grey columns of the anterior cornua. The great improvement, however, in such a short period as two months is more in favour of peripheral than central disease.

Vulpian also describes a case of ulnar paralysis coming on during convalescence from typhoid in a man aged 58. The patient first experienced a sensation of numbness in the little and ring fingers, and along the corresponding edge of the left hand, together with slight pain in the pulp of these two digits. This was followed by progressive feebleness and wasting of the muscles, and by anæsthesia of the skin supplied by the ulnar nerve.

Another of Vulpian's patients on the second day of convalescence from typhoid complained of pains in the right arm and shoulder. The pains were severe enough to prevent sleep. At the end of a week the limb became numb and weak. During the third week the pains gradually disappeared, but the weakness persisted, and was most marked in the deltoid, this muscle becoming manifestly atrophied. The scapular muscles were also wasted, and the right arm was thinner than the left. When examined three months later the deltoid was considerably atrophied, and did not respond to either the faradic or galvanic current. The muscles of the arm and forearm, however, had regained some power.

Vulpian mentions also three cases of atrophic paralysis affecting the muscles of the lower limbs. In one of these cases, a man, aged 23, at the decline of a severe attack of typhoid, suffered from severe pains, which came suddenly, first in one leg and then the other. The pains lessened in severity during the second week, but there was a progressive loss of power in the lower limbs, till the patient was unable to stand upright. The antero lateral region of the legs was chiefly affected, and the right more than the left leg. The right foot drooped, and the patient found it very difficult to flex the foot or extend the toes There were constant boring, rarely lancinating, pains in the lower limbs. A month after the onset of pain the right leg showed distinct muscular atrophy, and the extensor communis digitorum, and the extensor proprius pollicis responded but feebly to electricity. Five months later, in spite of treatment with the faradic current, the weakness and wasting of the leg muscles were unaltered, and there were still pains in the feet and lower part of the legs.

We now come to the very important paper by Pitres and Vaillard, who were the first to prove by careful microscopical observations that peripheral neuritis may be present in typhoid fever, and even apart from obvious manifestations of its presence during life. In this paper they relate two typical cases of ulnar paralysis occurring during the convalescent period of typhoid fever.

In one case a man, three days after an attack of typhoid lasting thirty days, was seized with acute lancinating pains in the pulp of the little finger of the left hand, which extended towards the wrist; these pains, constantly exaggerated by crises, were sufficiently severe to deprive the patient of sleep. There was also a sensation of numbness and tingling in the little and ring fingers, which was quickly followed by anæsthesia, and by an atrophic paralysis of the muscles in the territory supplied by the ulnar nerve. Trophic changes in the nails and skin were absent, but the left hand was always colder than the right, and became bluish on exposure to the air. In the seventh week from the onset of these symptoms the patient began to suffer from lancinating pains in the thumb and index finger, and these parts showed diminution of the cutaneous sensibility. This sensory disturbance indicated an affection of the radial nerve, but the power and electric contractions of the muscles supplied by this nerve did not become impaired.

The second case closely resembled the first one, with the exception that numbness and tingling preceded, instead of following, the lancinating pains.

Pitres and Vaillard then give the results of a histological examination of certain nerves removed from the bodies of four patients who died during the pyrexial period of typhoid. No symptoms were observed during life which could be ascribed to disease of the peripheral nerves, and yet changes significant of parenchymatous neuritis were found to a marked degree in three of the four cases.

In the first case, a man aged 23, who died on the 16th day of the disease, the following nerves were found altered: Branches of the internal cutaneous and musculo-cutaneous nerves to the forearm, some of the terminal branches of the ulnar nerve, and muscular branches to the pectoralis major. The changes were more pronounced in the nerves on the right than on the left side.

In the second case the patient died on the 36th day, and marked changes were found in the nerve branches to the dorsum of the left big toe, in the left peroneal, and the right anterior tibial nerves. Nerve fibres on the back of the right hand, and the trunk of the right ulnar were also affected.

In the third case the patient died from perforation of the intestine on the 24th day, and in the left peroneal, the right anterior tibial, and the right internal saphenous nerves the majority of the fibres were almost entirely destroyed.

The only other post-mortem evidence of peripheral neuritis in typhoid fever with which we are acquainted, is that afforded by two cases recorded by Oppenheim and Siemerling. In one case death occurred in the middle, in the other at the end of the second week of the fever. In the former case parenchymatous degeneration of the great saphenous and peroneal nerves was found ; in the latter, a branch of a cutaneous nerve on the dorsum of the right foot showed complete degeneration of many of its fibres, while the peroneal nerve was in a condition of partial degeneration.

Alexander remarks on the rarity of extensive and permanent paralysis of the extremities as complications or sequelæ of typhoid. He says that during ten years and a half in the Medical Clinic of Breslau, not a single case of paralysis occurred amongst 390 typhoid patients. He then relates the following case of paraplegia. A female, aged 20, had a very severe attack of typhoid fever; about seven or eight weeks from the onset of the disease on trying to get out of bed she was unable to stand. This was at first attributed to general weakness, but the weakness in the lower limbs increased ; she was scarcely able to move her legs in bed, and when supported in the erect posture she could not feel the floor. She complained also of pains in the legs, and had some difficulty in passing water. She suffered, too, from headache, and vertigo, and there was some weakness of the mental functions.

On examination, a month later, the temperature was normal, but the pulse very rapid (132). The voice was weak and husky, and there was paresis of the left vocal cord. The lower limbs were almost completely paralysed, slight flexion of the hip and knee being the only voluntary movements ; the muscles were greatly atrophied and gave the reaction of degeneration to electricity. The knee-jerks were lost, the abdominal reflex was normal, but a true plantar reflex was not obtainable. All forms of cutaneous sensibility were normal, and the nerves of the lower limbs were not tender to pressure. On making passive movements of the foot and toes, the sense of position was found to be impaired.

The weakness of the left vocal cord quickly passed away, but the other symptoms persisted for some time. After three months a slight improvement in the movements of the legs was observed, and a few weeks later the patient was able to stand without support, but the muscles gave no response to the faradic current. In about a year from the onset of the paralysis the right knee-jerk returned, but the left was still absent. The movements of the toes were feeble, muscular atrophy was considerable, but the muscles did not respond to electricity. Six months later the muscles had almost regained their normal bulk and electrical reactions.

Three cases of unilateral ulnar paralysis during convalescence from typhoid are recorded by Dr. Handford. In the first case numbness, anæsthesia, and paralysis of the muscles in the territory of the ulnar nerve followed a very severe attack of typhoid of about seven weeks' duration. During the few weeks the patient was under observation the condition of the hand improved to some extent, but "he was lost sight of before the restoration of motion and sensation was complete."

In the second case left ulnar paralysis came on during the third week of a moderately severe attack of enteric fever. The patient first "complained of *stiffness* of the ring and little fingers of the left hand." This was followed by anæsthesia and paralysis of parts supplied by the ulnar nerve. At the end of three months the patient had considerably improved, and eventually he got quite well.

The third case refers to a boy, aged 15, who, during a second relapse of typhoid, began to suffer from pain in the little and ring fingers of the right hand. This was followed by anæsthesia and an atrophic paralysis of the muscles supplied by the ulnar nerve. There was "marked tenderness on grasping the muscles of the little finger or of the ball of the thumb, and on pressing the arm along the course of the ulnar nerve nearly as far as the axilla." Two months later Dr. Handford says, "the power of movement has not improved much, though sensation has largely returned." The little and ring fingers remained in a marked "claw hammer" position, from the paralysis of the interossei and two inner lumbricales. "Muscular wasting is still very evident."

In a case recorded by Archer, paralysis of the right arm developed during the third week of an attack of enteric fever, at a time when the general symptoms were very severe. At first there was numbness of the right arm, then weakness and wasting of the muscles, associated at one time with great pain and heat of the limb. Subsequently the condition improved, but "the limb had not entirely recovered its functions when the patient was discharged in the tenth week."

Dr. F. R. Humphreys mentioned at a meeting of the Hunterian Society the case of a little girl who suffered in September, 1888, from a severe attack of typhoid fever. In the fourth week the abdominal reflexes were found to be greatly exaggerated, and to be accompanied by pain and flexion of the legs. In November, though able to sit up in bed, she could not lift either leg, and was unable to walk. The muscles of the lower limbs gave no reaction to the faradic current. In a short time the right leg regained power, but the abdominal muscles on the left side were noticed to "belly out" when the child cried or moved, and the weakness was especially conspicuous in the external oblique muscle. In February, 1889, that is, about five months subsequent to the attack of typhoid, it was found that all the muscles except the left tibialis anticus, the abductors of the thigh and the abdominal muscles reacted to the faradic current, and the child walked fairly well.

Madame Dejerine-Klumpke publishes a case in which paresis of the quadriceps femoris and of the tibialis anticus on the left side followed a slight attack of typhoid. There was no appreciable atrophy of these muscles; sensation was normal; the knee-jerks were lost.

George Ross has published two examples of paralysis after typhoid. In one case, four days after convalescence had commenced, the temperature rose to 102° F. and the patient began to suffer from severe pains in the legs, and there was much tenderness of the skin and muscles. The knees and thighs were rigidly flexed, and any attempt to straighten them was accompanied by pain. All the movements of the lower limbs were feeble, and the electrical reactions of the muscles much impaired. There was no anæsthesia, the superficial reflexes were diminished, the sphincters were unaffected.

Three months later the legs were gradually extended by means of weights and pulleys until they became quite straight. During this time motor power slowly returned and pain quite disappeared, and at the end of four months from the onset of the nervous phenomena, the patient had completely recovered.

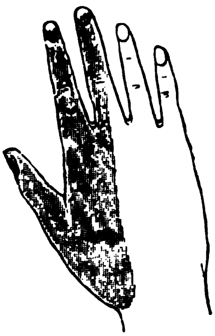
In the other case, a boy, at the end of a mild attack of typhoid, lasting four weeks, was observed to speak with effort, and with a nasal intonation. This was followed by a sensation of numbness in the ends of the fingers and in all the limbs, which became partially paralysed; the hand and arm were also raised with great difficulty, and the grasp was very feeble. Tactile sensation was good; there was no pain in the extremities; the knee-jerks were absent; the eyes were normal. The velum palati was relaxed and insensitive. After treatment with galvanism and strychnia for several weeks, the limbs had improved in power and nutrition to a moderate extent, and the voice was less nasal.

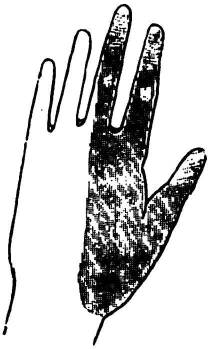
Dr. G. Ross thinks that diphtheria can be excluded with certainty from this case.

Of the few examples of paralysis succeeding typhoid fever that have come under my own observation, the most noteworthy case, and the one in which the symptoms pointed most distinctly to a multiple neuritis, was that of a girl under the care of Dr. Dreschfeld, to whom I am indebted for the following notes :—

Jane W., aged 18, was admitted to the Manchester Royal Infirmary, May, 1888. About eight months previously she had passed through an attack of typhoid fever of average duration and severity. During the fever she suddenly found one day that she was unable to straighten out her elbows, wrists, and knees, and the rigidity persisted. On admission to the Infirmary, there was much wasting of all the muscles of the limbs. In the lower extremities the muscular atrophy was especially conspicuous on the front of the thighs and on the outer part of the legs. The knees were semiflexed, and could not be fully extended, owing to the contractures of the flexor muscles. There was drooping of the great toes, and also of the other toes, though to a less degree. The knee-jerks were somewhat variable; as a rule they could only be elicited with difficulty, but sometimes they appeared to be unduly irritable. The plantar reflexes were absent, and there was no ankle clonus.

As to the upper limbs, the elbows were semiflexed, and could not be extended, owing to the rigidity of the flexors. The right forearm was more wasted than the left one, and presented distinct hollows on the anterior and posterior surfaces. There was also great atrophy of the thenar and hypothenar eminences, and moderate atrophy of the interosseous muscles, more marked in the right than in the left hand. The patient was unable to oppose the thumb against the end of the little finger, and indeed all the movements of the hands and fingers were feebly performed. There was almost complete anæsthesia over the right thumb, the index finger, and the radial side of the middle finger; the anæsthesia on the dorsal aspect implicating also the ulnar side of the two terminal phalanges of the middle finger. The cutaneous sensibility of the left thumb was also diminished. At the end of a month the





movements and nutrition of the upper limbs had improved, but muscular atrophy was still considerable. The patient was put under chloroform, but the contractures at elbows and knees could not be overcome. She remained in hospital till the end of June, when the limbs had regained some of their volume and power, but the contractures and anæsthesia persisted. Dr. Dreschfeld informs me that ultimately the patient made a complete recovery.

Another case was that of a man aged 42, who came to the outpatient department of the Royal Infirmary about two years ago. He stated that his legs had been weak and numb since an attack of typhoid fever, for which he had recently been treated in the Monsall Fever Hospital. On examination there was partial loss of the cutaneous sensibility, chiefly to pain in the feet and lower part of the legs, and patient said that his feet felt sore, and that he suffered from pains in the legs after walking. The movements in the toes and feet were feeble, and all the muscles below the knee presented a moderate degree of wasting. The calf muscles appeared to be especially weak; in walking the man leaned further back than normal; he could balance himself on his heels for a time, but was unable to stand on his toes. The knee-jerks were exaggerated. There was no suspicion of syphilis, alcohol, or other cause than that of typhoid fever.

The preceding observations furnish abundant evidence that paralysis involving the whole or a portion of a limb sometimes occurs either during or subsequent to the pyrexial period of typhoid fever, and they illustrate the variations in the distribution of the paralysis. In a large proportion of the cases certain features are conspicuously prominent, and throw much light on the nature and situation of the lesion.

(1) The paralysis is usually partial in extent and degree. It may affect the whole of both lower limbs, or one limb either upper or lower, but as a rule it is limited to certain muscles or groups of muscles, and often is strictly confined to the territory of a particular nerve.

(2) The muscles are wasted as well as weak, and when tested with electricity show the partial or complete reaction of degeneration. In a few cases contractures of muscles are mentioned, but it is to be observed that they were found not in a well nourished more or less rigid limb, but in one where some at least of the muscles were distinctly atrophied.

(3) The paralysis is almost invariably preceded or accompanied by some form of sensory disturbance. Thus the patient suffers from intense pain, continuous, remittent, fixed or radiating, or he complains of numbness and tingling in the extremity, which subsequently becomes paralysed. Anæsthesia, partial or complete, is of frequent occurrence, and like the paralysis which it usually precedes, may be limited to the territory of a particular nerve.

(4) During the course of typhoid fever, the knee-jerks, as pointed out by Angel Money, are commonly exaggerated, and there may be ankle clonus. Gowers refers to excessive knee jerk and foot clonus in association with paraplegic weakness in patients seen some months after the fever; and in the case of the man under my own care referred to above, there was exaggeration of the knee-jerks, together with paraplegic weakness and anæsthesia. But, as a rule, judging from the reports in which the reflexes are mentioned, paralysis of the lower limbs is associated with diminution or loss of the knee-jerks.

The peripheral paralyses, then, which occur in association with typhoid fever are, for the most part, of the atrophic variety—that is, the muscles not only become weak, but they undergo a progressive atrophy, and give the reaction of degeneration to electricity. An atrophic paralysis testifies to a lesion situated in some part of the lower segment of the motor tract—that is, the motor nerve fibres or their nuclei of origin. Now while it is probable that in a few cases the muscular atrophies which follow typhoid fever depend on an anterior poliomyelitis, and that a condition similar to that of infantile paralysis is produced, the presence of sensory disturbance in the vast majority of cases shows that the lesion, if in the cord at all, is not limited to the anterior horns or involves both the anterior and posterior roots, or the mixed peripheral nerves. The absence of spinal tenderness, of girdle pains and of disturbance of the sphincters, speaks much against an affection of the spinal cord or its roots, while the initial sensory disturbance, succeeded by a limited paralysis having a slow progressive march up to a certain degree, which varies according to the severity of the case, the paralysis then slowly receding and ultimately, as a rule, completely disappearing, are points strongly in favour of an affection of the peripheral nerves. Sensory disturbance is indeed one of the most distinctive features of typhoid paralysis; the pains are frequently severe enough to prevent sleep, and loss of skin sensibility may be associated with hyperæsthesia of the muscles, leading to painful contractures, just as in cases of alcoholic paralysis. Hyperæsthesia of the skin or muscles and neuralgiform pains may exist alone and not be followed by any detectable weakness or wasting of muscular tissue. Landouzy relates the case of a man who from the first days of his attack of typhoid fever suffered from such intense pain at the level of the right sciatic notch that he gave himself hypodermic injections of morphia. This was succeeded by severe pain radiating along the branches of both sciatic nerves; at the same time there were severe cramps in the thighs and calves. Towards the thirtieth day, when he lay on his right thigh, contact with the bed was not felt, although great pain was produced in the limb. This tract of "anæsthesia dolorosa" was found to correspond roughly with the distribution of the external cutaneous nerve. Eight years later the anæsthesia still existed, though to a less degree, but the painful phenomena had disappeared.

As a parallel to cases in which pain and hyperæsthesia are prominent features may be mentioned cases characterised by excessive muscular contractions. Of examples of the latter none are more remarkable than those recorded by F. A. Aran, in *L'Union Médicale*, July 19, 1855. He mentions eleven cases in which muscular contractions occurred towards the termination of attacks of typhoid fever, and one case in which the contractions coincided with the commencement of the fever. They were frequently preceded by formication, prickings and numbness in the extremities, and by pain in the joints. The immediate seizure was accompanied by an intense feeling of anxiety and distress. The contractions affected both the upper and the lower limbs, and many of the muscles exhibited almost incessant fibrillary contractions. When the entire upper extremity was attacked the forearm was bent upon the arm and lay upon the thorax, while the fingers were doubled up on the palm of the hand. The patient was unable to extend the limb, but by gradual manipulation an artificial extension could be obtained, and this gave the patient relief, though the parts resumed their morbid position when left to themselves. When the lower extremities were attacked the legs became extended, the calves hard and painful, and the toes flexed. In four cases the muscles of the trunk were affected, and opisthotonus was produced, during which the patient was unable to execute any movement. During the seizures the contracted muscles were the seat of great pain. The attacks lasted from a quarter of an hour to three hours, and recurred from two to ten times daily for several days. After the cessation of the attacks the fever ran its ordinary course without any residuary affection beyond an occasional numbness of the affected parts. Three of the patients died, but this result was attributed by Aran to the severity of the fever, and not to the tetanic affection. Dr. Aran was at a loss to account for the phenomena. Baths appeared to relieve the patients, and in one case of opisthotonus the administration of chloroform was of benefit.

Having relegated, with a high degree of probability, the majority of post typhoidal paralyses to lesions of the peripheral nerves, we have still to enquire whether the neuritis is the result of accidental circumstances attending the fever, or whether it may be regarded as a direct result of the typhoid virus. Now, in some of my collected cases, the paralysis, both sensory and motor, is limited to the parts supplied by a particular nerve, and in these it may be supposed that the neuritis is caused, not by a poison brought to the nerve by the blood or lymph, but by a local injury resulting from pressure or stretching of the affected nerve at a time when its nutrition is defective, owing to the general weakness left by the fever. The theory of local injury, for the explanation of many cases, at least, is adopted by Dr. Gowers, but without denying that it may be a potent factor in some cases there are not wanting considerations which tend to show that the cause of the neuritis, in most cases, is to be found in some kind of poison conveyed by means of the blood to the affected nerves; for even when the chief functional disabilities are limited to one nerve territory, there are frequently slight indications of a more widely distributed disorder. These remarks may be illustrated by a brief reference to the three cases of ulnar paralysis described by Dr. Handford, which the author seems inclined to attribute to a local injury. "In the first and last cases," says Dr. Handford, "the patients were accustomed to lie 'curled up,' with the elbows sharply flexed, for days together, and each chiefly on the side eventually affected. It is quite possible that thus a neuritis from over-extension was set up, and that the earlier numbness and tingling were disregarded during the delirious condition. And further, I can hardly conceive a more favourable condition of tissue malnutrition than these two cases presented." But with regard to his second case, the author says: "His attack of fever was not much above the average severity; he had no throat affection, no great loss of flesh, and lay always on his back, with the arms generally extended." So that it is difficult to see how the theory of overstretching by persistent flexion would apply in this case, and even in one of the two severe cases, in addition to the ulnar paralysis, other notable symptoms were present which demand explanation, and which could not be readily accounted for by the theory of local injury. "For some weeks past," says Dr. Handford, "there has been *tenderness of the toes* of both feet, so that the toe nails could not be cut on account of the pain it caused in the nail bed and in the pulp at the end of the toes." "In three other cases recently," he continues, "I have met with this condition of pain in the toes, and in one of them in the arms also. In one instance the tenderness of the feet was so great that they had to be protected from the pressure of the bedclothes by a cradle. But in none of them was it followed by muscular wasting or definite loss of sensation, so far as I could detect." In other words, in these cases there was evidence of profound irritation of the cutaneous nerves of the lower extremities, but which had not produced destruction of nerve fibres, and therefore was not attended by either sensory or motor paralysis.

In addition to the group of paralyses described in the preceding pages there are a few observations showing that occasionally an acute paralysis of rapidly ascending course, and frequently terminating fatally, may develop during the course of, or immediately after, an attack of typhoid fever. In his exhaustive account of Landry's paralysis, Dr. Ross has so fully discussed the relation of this disease to peripheral neuritis that no further reference to the subject is here needed.

From a consideration of the preceding observations we are inclined to think that a multiple neuritis, initiated by the virus of typhoid fever, and frequently present in this disease, is not an unreasonable assumption. In one set of cases we may suppose that the action of the poison on the nerves is too slight to give rise to outward manifestations, or that these are masked by the general symptoms of the fever. In another set of cases the toxic influence on the nerves appears to be revealed by a series of irritative phenomena, such as neuralgic pains, cutaneous and muscular hyperæsthesias, exaggerated reflexes, and cramps and contractures of various muscles; while in a third group of cases the presence of an atrophic paralysis, its distribution, progress, and associations leave little doubt on the mind of the observer that he has to deal with a genuine parenchymatous neuritis, and frequently with one of wide distribution throughout the body.

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(c) Typhus Fever.

Judging from the scanty literature of the subject, peripheral paralyses are much rarer complications or sequelæ of typhus than of typhoid fever.

Murchison[*] says: "Occasionally the muscles of one limb or individual muscles, such as the deltoid, are paralysed. These muscles after a time become shrivelled, and if some be more atrophied than others, club foot and other distortions may result. These attacks of paralysis are often preceded by severe pain or pricking sensations, and are accompanied by numbness, complete anæsthesia or hyperæsthesia of the affected part. The pathology of these attacks of paralysis is obscure, but they are probably due to arterial thrombosis of the central organs of the nervous system or of individual nerves." In the following case reported by Murchison the distribution of the paralysis, the character of the sensory phenomena, together with the normal condition of the sphincters, suggest a multiple neuritis rather than disease of the spinal cord.

Case 21.—"Typhus,[†] followed by general paralysis and anasarca and remarkably slow pulse."—A gentleman, aged twenty-seven years, caught typhus in the beginning of May, 1855. "The attack was severe, being characterised by a copious rash, great and protracted delirium, involuntary stools, and inability to swallow. The treatment consisted in stimulants and numerous blisters to neck and behind the ears. On recovery from the fever the blistered surfaces took on unhealthy action and sloughed. Patient remained very prostrate and became greatly emaciated, while legs were œdematous and abdomen was thought to contain fluid. It is uncertain whether or not urine contained albumen. On application of sulphate of copper to ulcerated surfaces the profuse discharge suddenly ceased, but the day after patient had an epileptiform fit which lasted about two minutes. For this he was purged and kept on low diet, while the dropsy was treated with squills and nitric ether. Under this treatment he became much weaker and lost flesh; eyesight became impaired, and for a fortnight he was unable to read large type or even to distinguish large objects plainly. About the same time he began to complain of pricking sensations, first in toes and afterwards in fingers, and inability to perform finer duties of fingers, such as buttoning his shirt. These pricking sensations gradually extended up limbs, and were followed by numbness and loss of power, ending with complete paralysis of both motion and sensation, although slightest handling of calves of legs excited most exquisite pain. Tongue was also œdematous, and was seat of pricking sensations, while muscles of deglutition were paralysed, so that swallowing could only be performed slowly and required attention of mind. The integuments of abdomen were devoid of sensation, but there was no paralysis of rectum or bladder. Mind was unimpaired." In August, 1855, after taking a cold splash bath he acquired some motion in hands, and by end of a month he was able to stand; in two weeks more he could walk about. He progressed favourably, and in a few months he was able to follow the duties of his profession as army surgeon.

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[*] Murchison, *loc. cit.*, p. 206.

[†] Murchison, *loc. cit.*, p. 201.

One day, three years later, "after seeing some hospital patients, he suddenly felt his heart working in a strange manner, thumping slowly, and at same time he experienced a feeling of giddiness." His pulse was found to be barely 40, and during the next fourteen years it varied between 34 and 36. There was some dyspnœa on exertion, but the heart appeared to be normal, and no cause could be found to account for the extreme slowness of the pulse.

Murchison also refers to the case of a female, aged 48, in whom temporary facial paralysis showed itself on the seventeenth day; and Gairdner to a case in which facial paralysis was observed on the tenth day of typhus fever.

A case of paralysis of all the muscles supplied by the right musculo-spiral nerve is reported by Bernhardt* which supervened suddenly during convalescence from typhus fever. The faradic contractility of the paralysed muscles was nearly abolished, and the galvanic contractility was diminished. The patient complained of tingling in the hand and fingers, but the cutaneous sensibility to pin pricks and temperature appeared to be normal. The patient died of laryngeal perichondritis and pneumonia five weeks after the appearance of the paralysis, and at the autopsy the musculo-spiral nerve was found swollen, and of a violet grey colour, at the point where it turns round the humerus; below this point the trunk of the nerve and its branches were profoundly altered, the axis cylinders being for the most part destroyed and replaced by bands of connective tissue, which were separated by long threads of granules, composed no doubt of broken up myelin.

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* Bernhardt. "Zur Pathologie der Radialisparalysen."—*Arch. f. Psych.*, Bd. IV., 1874, p. 601.

The abrupt onset of the paralysis without appreciable cause, its occurrence during convalescence, and the severity of the symptoms, are points which make Bernhardt inclined to adopt the hypothesis of a local determination of infection by the virus of typhus rather than that of mechanical compression as the most likely explanation of the neuritis.

(*d*) RELAPSING FEVER.

Murchison says:—"Partial palsy, lasting for a few days or weeks after recovery, is occasionally noticed. Cormack mentions the case of a female, aged thirty-six, in whom loss of power in both deltoids continued for about ten days, after restoration to health in every other respect had taken place. In two (of 220) cases Douglas observed partial paralysis of the fore arms; in one it came on during the intervals between the attacks; in both cases the attack was sudden, with accompanying numbness, but with no head symptoms; the paralysis lasted for several weeks. Temporary paralysis of the upper and lower extremities was observed by Dr. Parry in several cases at Philadelphia in 1869-70. In a man, æt. 44, Tennent observed paralysis of the portio dura supervene six days after the second crisis."

MURCHISON. *Loc. cit.*, p. 382.
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(*e*) SMALLPOX AND CHICKENPOX.

Dr. Ross justly remarks that "The paralyses which supervene in the course of ordinary convalescence from smallpox are more numerous, more variable, and more grave than those which occur after the other exanthemata." The most common forms are undoubtedly of spinal origin, but there are a few cases in which the paralysis appears to depend on disease of the peripheral nerves. Thus, excluding the cases of acute ascending paralysis following variola, and tabulated by Dr. Ross (see pages 18—40), there are examples of local paralysis such as the case published by Vulpian and that by Joffroy. In Vulpian's case a man during convalescence from variola was seized with acute pains in both arms, and subsequently the muscles about the shoulders became paralysed and wasted. The faradic contractility was abolished in the deltoids, supra- and infra- spinati muscles, and the inferior portions of the pectorales majores; and the cutaneous sensibility was diminished over the region of the deltoids.

Joffroy's case was that of a woman, who, during convalescence from a severe attack of confluent smallpox, complained of violent pain in the left shoulder, and subsequently down the left arm. The deltoid, the triceps, the extensors of the hand and fingers, the supinator longus, and the small muscles of the hand became paralysed and atrophied, and failed to react to a strong faradic current. The patient died of pulmonary tuberculosis: no lesion was found in the cord on microscopical examination, but the left musculo-spiral and the left ulnar nerves presented marked changes, the axis cylinders and the myelin having disappeared from a large number of nerve fibres.

Dr. Gowers says: "In one case, which developed six weeks after varioloid, with pains in the limbs, atrophy of the arms and legs, etc., the purely neuritic nature of the morbid process was ascertained six months later when the patient died from pneumonia."

GUBLER. *Archiv. génér. de Méd.*, 1860.

LEROY D'ETIOLLES (RAOUL). "Du paralysies des membres inférieurs ou paraplégies."—Paris, 1856.

VULPIAN. *Arch. de Physiologie*, Tome V., 1873, p. 95.

JOFFROY. "De la névrite parenchymateuse spontanée."—*Arch. de Physiologie*, Tome VI., 1879, p. 177.

HENDERSON (G. C.). "Smallpox followed by ataxy."—*Lancet*, 1881, II., 1089.

GROSS. "De quelques accidents nerveux compliquant la variole et la variolide."—*Alger. Med.*, 1883, XI., 328, 353.

MABILLE. "Demence paralytique à la suite d'une variole."—*Ann. Méd.-Psych.*, 1883, 6 s., X., 225.

QUINQUAUD (E.). *Encéphale*, Paris, 1884, IV., 33—47.

WHIPHAM and MYERS (A. T.). "On some chronic nervous sequelæ of smallpox, especially affecting the speech."—*Transactions Clinical Society*, 1886, p. 164.

COMBEMALE (F.). "Contribution à l'étude des troubles (paralytiques et ataxiques) de la parole consécutifs à la variole; leur fréquence, leur pathogenie." [Contains a good bibliography relating to paralysis after variola.]

(*f*) Scarlet Fever.

No case is known to me in which paralysis occurring in connection with scarlet fever, could be attributed with any certainty to peripheral neuritis; and it is highly probable that recorded instances of more or less generalised paralysis, as, for example, the case published by Shepherd, depended on diphtheritic rather than scarlatinal poison. Bassette has reported the case of a child aged ten, in which paralysis of the upper and lower limbs, with atrophy and deformities, followed scarlet fever. The upper extremities recovered, but paralysis and atrophy persisted in the lower. He says that "the atrophic paralysis was in all probability the result of an inflammatory affection which attacked both nerves and cord, and was due to the action of the infection upon nerves and nerve centres."

Shepherd. "Paralysis after scarlet fever."—*The Medical Times and Gazette*, Vol. I., 1868, p. 144.

Cutter (E.). "Case of scarlatina, followed by paralysis of the tongue."—*Maryland Med. Jour.*, Balt., 1879-80, V., 171.

Fraser (D. M.). "The nervous sequelæ of scarlet fever.—*Pract.*, 1882, XXVIII. 431; XXIX., 22.

Schotten (E.). "Ein Fall von Tetanie nach Scharlach."—*Berl. klin. Woch.*, 1888, XXV., 273.

Loeb (M.). "Ueber einige seltenere Complicationen des Scharlachs (Peptonurie, Tetanie, Aphasie, Ataxie)."—*Arch. f. Kinderh.* Stuttg., 1889, X., 212.

Thomas. "Troubles nerveux post-scarlatineux."—*Rev. méd. de la Suisse Rom.*, 1891, XI., 761.

Bassette (M. J.). "The paralyses in children which occur after and during infectious disease."—*Journal Nerve and Mental Diseases*, 1892, p. 461.

(*g*) Measles.

About sixty cases of paralysis developing during the course of, or in early convalescence from, measles, are to be found recorded in medical literature. The large majority of these were of cerebral or spinal origin; and as regards the remaining cases of paralysis, some were probably due to a multiple neuritis, although a study of the clinical histories does not yield absolutely certain evidence of the existence of neuritis.

Ormerod has recorded three cases of muscular atrophy following measles, but these can scarcely be regarded as examples of peripheral neuritis, for the deep reflexes were preserved, and sensory phenomena were absent from first to last; and it is probable, as Ormerod suggests, that they belong to the group of myopathic atrophies.

Denarié states in his fifth conclusion (p. 98) that the most frequent form of paralysis that occurs in childhood is a paraplegia, unaccompanied by disturbance of sensation, by trophic troubles, by modifications of the tendon reflexes, or by disturbances of the bladder or rectum, and a paralysis which sooner or later is completely cured.

He mentions several cases of weakness and wasting of the lower limbs, in some of which the clinical description affords evidence of the possible presence of peripheral neuritis. The case of a little girl is recorded by Schepers, who after an attack of measles was unable to walk, and he states that the symptoms were more like those of acute ataxia than of paralysis. The patient made a complete recovery in about 15 days.

Annesley (A.). "Paraplégie suite de rougeole."—*Arch. de méd. et pharm. mil.* Paris, 1886, VIII., 386.

Bayle (L.). "Des paralysies consécutives á l'infection morbilleuse."—Paris, 1886, *Thesis.*

Perret (S.). "Des paralysies consécutives á la rougeole."—*Clin. méd de l'Hotel-Dieu de Lyon.* Paris, 1887, 455.

Négrié. "Un cas de paralysie ascendante aiguë consécutive á une rougeole."—*Journal de méd. de Bordeaux,* 1887-88, XVII., 22.

Fox (T. C.). "An affection of the nervous system during the exanthem stage of measles."—*Lancet,* 1887, I., 771.

Denarié (A.). "Des paralysies dans la rougeole."—Lyon, 1888, *Thesis.*

Allyn (H. B.). "Paralysis following measles."—*Medical News,* Philadelphia, 1891, LIX., 617. [This paper contains references to 41 cases.]

Carpenter (J. S.). "A case of paralysis following measles."—*Medical News,* Philadelphia, 1892, LX., 183.

(h) Cerebro-spinal Meningitis.

At a meeting of the Philadelphia Neurological Society, Dr. C. K. Mills made some remarks on the probable occurrence of multiple neuritis in this disease. He mentioned three cases in which the symptoms seemed to point to neuritis, involving certain portions of the body. In one case the patient complained of pain and soreness in his legs. There was tenderness over the nerve trunks, and muscular and cutaneous hyperæsthesia. The patient had also the equino-varus position of the feet so often seen in multiple neuritis. The knee-jerks were less active than normal. Dr. Mills thought that "in some cases of 'cerebro-spinal meningitis' the multiple neuritis might alone be present, and that in this way certain irregular cases might be accounted for." In Upson's case the ulnar nerve, which was the only one examined, showed parenchymatous and interstitial changes.

Mills (C. K.) "The probable occurrence of multiple neuritis in epidemic cerebro-spinal meningitis."—*Med. News,* Philadelphia, 1888, LII., 357.

Upson (H. S.) "On a case of multiple neuritis and cerebro-spinal meningitis."—*Journal of Nervous and Mental Disease,* New York, 1889, XVI., 351.

(i) INFLUENZA.

Affections of the peripheral nerves were exceedingly common during the last two epidemics (1889-90, 1890-91) of influenza. Isolated neuritis of almost every peripheral nerve in the limbs and of almost every cranial nerve has been recorded, while multiple neuritis, though less common, was occasionally observed. The evidence as to the existence of a genuine attack of influenza before the occurrence of neuritis is not always satisfactory, for it seems probable that nerve symptoms have followed slight or abnormal attacks of influenza—attacks which are liable to be imperfectly observed—more frequently than typical seizures of the disease. The paucity of accurate observations renders a full discussion of the subject undesirable at present, but it will be useful to collect together a few of the more important cases as an aid to future work on the connection between influenza and multiple neuritis.

Brosset relates the case of a woman, aged 34, who, a fortnight after the onset of an attack of influenza, had pricking sensations in the left hand and lancinating pains in the soft parts of it. Four months later there were analogous pains in the right hand. Then pains in the feet and difficulty in walking ensued, but no inco-ordination of movement. Her condition eleven months after the attack of influenza was as follows: There was considerable loss of motor power with some rigidity, muscular wasting and a thinning of the skin, as well as diminished reflexes and pains in the soft parts of both hands and feet. The patient suffered throughout her illness from profuse perspiration.

A man, aged 39, under the care of Homen, suffered during convalescence from influenza, from numbness and weakness of the right arm, and a few weeks later of the right leg. Three weeks afterwards the left extremities were implicated, the arm being attacked before the leg. The weakness increased during the next month, and then became stationary. The hands, feet, and legs were the seats of slight pains. When examined four months after the influenza, Homen found almost complete paralysis of the extremities, especially of the upper ones, and atrophy of the muscles which gave the reaction of degeneration.

The cutaneous sensibility was diminished in the arms and in the upper part of the trunk, and to a less degree in the lower limbs; painful sensations were delayed and the muscular sense was diminished, especially in the hands and feet. The knee-jerks were present, but no tendon reflexes could be obtained in the arms. Many of the nerve trunks were tender to pressure. The bladder and rectum were normal. Gradual improvement occurred under the influence of massage, electricity, and tonics.

Dr. Mills records an interesting case of neuritis with a myxœdemoid condition of the limbs. The patient, after a sharp attack of influenza, became extremely weak in the legs, and was scarcely able to drag herself along. In a few days her feet and legs began to swell and to be painful, and soon became of enormous size and exquisitely tender. She gradually improved, but when last seen by Dr. Mills she still had a condition of firm swelling, which did not pit on pressure, from her knees to her ankles, together with great tenderness on squeezing the feet or ankles, or in handling the nerves or muscles of the limbs.

In a case recorded by Thue, paresis of all the limbs, double facial paralysis with complete reaction of degeneration, diplopia, anomalies of taste and slight anæsthesia, gradually developed during convalescence from influenza. The diagnosis made was polyneuritis with affection of the 6th and 7th cranial nerves, possibly also of the glossopharyngeal and hypoglossal as well as of the nerves of the extremities.

Westphalen has recorded two cases of "multiple neuritis after influenza." In the first case a man, aged 29, a week after a slight attack of influenza, complained of tingling and numbness in the fingers and toes. About 8 days later his legs and arms became feeble, and he had difficulty in swallowing. There was no pyrexia. On examination, the motor power of the limbs was diminished, but the muscles and nerves responded to the faradic current. The patellar, biceps, and triceps reflexes were absent, while the abdominal and cremasteric were preserved. There was probably a slight weakness of the right facial muscles, and a moderate difficulty in swallowing. The patient slowly recovered, but the knee-jerks three months later were still absent.

In the second case, an attack of influenza was followed in eight days by urticaria and paralysis of the lower limbs. During the next two weeks the whole body became gradually paralysed, there were also double facial paralysis and difficulties in swallowing. At the same time the reflexes were abolished. The muscles became atrophied, and the reaction of degeneration was obtained in the biceps, deltoid, interossei, glutei, and muscles of the thigh, as well as in the left facial muscles. There was muscular hyperæsthesia and tenderness on pressure over the nerve trunks. Beyond paræsthesia there were no sensory disorders. Ultimately there was almost complete recovery.

If Westphalen's cases are instances of multiple neuritis, they are remarkable for the absence of anæsthesia; and indeed the only indications of any disorder of sensation consisted in the presence of numbness and tingling at the onset in the first case, and of increased sensitiveness of nerves and muscles in the second case.

A still more remarkable example of a purely motor paralysis following influenza was shown by Dr. Buzzard at a recent meeting of the Neurological Society, and Dr. Buzzard has very kindly given me permission to mention this case, which I trust he will shortly publish *in extenso*. It was that of a man, aged 46, who shortly after a slight attack of the character of influenza suffered from pains in the legs, and some numbness of the feet. He soon began to totter in walking, and in a few months was unable to support himself. His upper limbs also became weak, but he had never any pains, twitching or tremor in them. Alcoholism and syphilis could be excluded. When examined by Dr. Buzzard about 13 months after the onset of the illness, there was a condition of almost universal paralysis. The masseters were weak; the voice was weak and hoarse; the four limbs were quite flaccid and helpless, the lower ones were absolutely paralysed, and the only movement retained in the upper limbs was the slightest possible power to lift the shoulders; the intercostal muscles acted, but the diaphragm was weak and the patient was unable to bend or straighten his back. The muscles were much atrophied, and no reaction to the strongest faradic current could be obtained in any muscle of the trunk or limbs. All the reflexes, both superficial and deep, were abolished. Cutaneous sensibility was unaffected; fibrillary muscular contractions were never seen. For a few days after admission to hospital the condition of the patient was critical, owing to his difficulty in breathing, but a very slow improvement soon began, and in a year's time he could move the arms pretty well, but the knee-jerks were still absent, and there was no evidence of the action of the diaphragm.

This case raises the question whether a purely motor wide-spread paralysis may be due to multiple neuritis. The motor tracts in the spinal cord may be diseased, while the sensory ones remain normal. Does a similar selection ever obtain in peripheral neuritis? In the common type of lead paralysis motor nerves are alone implicated, and may not the motor fibres of a large number of peripheral nerves be attacked by certain poisons, while the sensory fibres are spared? It is difficult to give a positive answer to this question, which suggests itself again and again with regard to the more generalised forms of motor paralysis from toxic causes. There appears to be no post-mortem evidence in favour of the central origin of these cases, and certainly the favourable course they usually run, together with the ultimate complete restoration of muscular nutrition and strength, point rather to an implication of the peripheral nerves.

Bergmeister (O.). "Ueber die Beziehungen der Influenza zum Sehorgan."—*Wien. klin. Wochenschr.*, 1890. (In one case paresis of accommodation; in two cases, amblyopia, probably from atrophy of optic papillæ following neuritis.

Uhthoff (W.). "Ueber einige Fälle von doppelseitiger Accommodations—lähmung in Folge der Influenza in dem einen dieser Fälle complicirt mit Ophthalmoplegia externa."—*Deutsche med. Wochenschr.*, 1890.

Laache (S.). "Facial paralyse nach der Influenza."—*Norsk. Mag.*, 1890, p. 841.

Thue (N. J.). "Polyneuritis mit doppelseitiger facial paralyse nach der Influenza." *Norsk. Mag.*, 1890, p. 851.

Draper. "The complications and sequelæ of Influenza."—*New York Medical Record*, 1890.

Westphalen (H.). "Neuritis multiplex nach Influenza."—*Petersb. med. W.*, 1890.

Bidon (H.). "Étude clinique de l'action exercie par la grippe de 1889-90 sur le système nerveux."—*Revue de Médecine*, 1890.

Revilliod (L.). "Des formes nerveuses de la grippe."—*Revue méd. de la Suisse Romande*, 1890. (He considers that the limb pains of influenza are distinguished from those of rheumatism by the very marked hyperæsthesia of skin and bones with which they are associated. The disturbances of motility are very variable, and comprise paraplegias, spastic and tetanic conditions, together with bulbar symptoms.)

Ferguson (J.). "A case of neuritis of the viscera."—*The Alienist and Neurologist*, 1890.

Homén. "Neuriter efter influenza."—*Finsk. läk. han.*, 1890; also *Neur. Centralb.*, 1891.

Church. "Multiple neuritis."—*Journal of the American Medical Association*, 1890.

Brosset. "A case of peripheral neuritis after influenza."—*Lyon Médical*, 1891.

Havage. "Note sur un case de polynévrite infectieuse aigué."—*Rev. de. Med.*, 1891. (The evidence as to influenza in this case is not quite satisfactory.)

Liegevis (Ch.). "Parésie des membres inférieurs et de la vessie dans l'influenza."—*Le Prog. Médical*, 1891.

Raoult (A.). "La grippe pendant l'hiver 1891-1892.—*Le Prog. Médical*, 1892.

Mills (C. K.). "The nervous and mental phenomena and sequelæ of influenza."—*Trans. of Phil. Medical Society*, 1892.

Althaus. "Influenza."—London, 1892, p. 165-195.

(*k*) ERYSIPELAS.

Paralysis is a rare sequela of erysipelas. Gowers mentions a case published by Feith, in which "paralysis of the soft palate and of the posterior crico-arytenoids followed facial erysipelas and one-sided pneumonia. The paralysis of the larynx necessitated tracheotomy, and appears to have been permanent."

Leu reports a case of facial erysipelas in a man who, two days after his temperature had become normal, began to suffer from paræsthesia in his legs, and pain in the muscles and along the nerve trunks, these parts being also tender on pressure. A little later the upper limbs were affected in a similar manner. There was diminution and then absence of the patellar reflex, together with disturbances in the electrical irritability of the nerves and muscles. The bladder and rectum were not affected.

The following phenomena were also observed—albuminuria, glycosuria, acceleration of the pulse, enlargement of the spleen, shortness of breath, glossy skin, hyperidrosis, herpes, and a miliary-like eruption on the trunk.

The acute symptoms, which lasted a fortnight, were treated with salicylate of soda, antipyrin, and opiates. Then after three months treatment with electricity and massage there was almost complete recovery.

Leu considered the case an example of the sensory form of multiple neuritis as described by Leyden.

Pal has recorded in his thesis on multiple neuritis the case of a woman aged thirty-four who, when examined two months after an attack of erysipelas of the trunk which had followed a surgical operation, was found to have the following symptoms: There was paralysis with much flabbiness and wasting of the muscles of the lower limbs, with the exception of the flexors of the knees, which were in a state of rigid contraction. The knee-jerks were abolished. The muscles of the upper limbs were also greatly wasted, the extensors of the forearm and the small muscles of the hands having especially suffered. There were tenderness of the nerve trunks to pressure and diminished cutaneous sensibility in the neighbourhood of the toes. There was complete loss of memory. The patient, after a seven months' treatment by massage had almost completely recovered. It was ascertained that she had had a similar attack of paralysis nine years previously, after erysipelas of the face.

In a case of severe erysipelas of the right leg, terminating fatally, Pal examined the peripheral nerves and found them quite healthy; and it is probable that in both Leu and Pal's cases the multiple neuritis is to be explained by the action of some poison circulating in the blood or lymph, and not by a direct spread of erysipelatous inflammation from the superficial parts to the nerves.

FEITH. *Berlin Klin. Wochenschrift*, 1874, quoted by Gower's *Dis. of N. S.*, Vol. II., p. 826.

BRIEGER. "Ein Fall von Parese beider Ober- und Unterextremitaten im Anschluss an Erysipelas faciei."—*Charité-Annalen*, 1885, und *Neurol. Centralblatt*, 1885, p. 444.

BROWNING (W.). "Multiple neuritis as a sequel of erysipelas."—*Brooklyn Med. and Surg. Journal*, 1888, I., 11.

LEU. "Ein Fall von infectioser multiple Neuritis nach Erysipelas faciei."—*Charité-Annalen*, XV., 1890, p. 274; and *Neurol. Centralblatt*, 1890, p. 728.

PAL. "Über multiple neuritis." Wien, 1891.

(*l*) SEPTICÆMIA.

Symptoms indicative of multiple neuritis occasionally follow the blood poisoned state, which occurs as a result of some injury or local inflammation.

Mary S., aged 58, came to my outpatient room in March, 1890. She stated that about 7 weeks ago she had a "gathering" at the end of the left middle finger; this was followed by blood poisoning and erysipelas, and the arm was swollen below the elbow. The finger and arm got well in 3 or 4 weeks, but shortly afterwards—that is, about 10 days before coming to the Infirmary—the fingers of the left hand became numb, and "the use went out of it." About the same time or perhaps a little later, the right hand also became numb and weak; then the weakness extended to the arms and legs. When she walked she felt as if a rope were tied round the ankles, and she found it "difficult to step out." It was "hard work getting up stairs," she could not bear the weight of her body on one leg while lifting up the other leg. She felt as if walking on a sponge. She never had pain, nor cramps in either arms or legs. On examination there was partial anæsthesia to pin-prick and touch over the hands and forearms; the diminution in cutaneous sensibility being most marked on the left side. The grasp of the hands was very weak, and all the movements of the fingers and thumb were feeble. The small muscles of the hand were moderately wasted. The end of the left middle finger was red and slightly swollen, and the terminal phalangeal joint anchylosed. The knee-jerks were diminished; and the muscles below the knees, especially on the anterior aspect of the legs, were weak and slightly wasted.

There was no reason to suspect alcohol or syphilis, and no history of sore throat or of exposure to diphtheria.

Under the influence of tonics and massage the symptoms gradually disappeared, and in three months the patient was almost quite well.

In this case there was an interval of five or six weeks between the first appearance of the gathering on the finger and the symptoms of neuritis. In all probability, too, a few days intervened beween the subsidence of the local inflammation and the first indications of neuritis. This interval has been observed in other cases. Thus Roth mentions the case of a man who "thirteen days after a stab-wound beneath the clavicle, which healed well, had an attack of parotitis with facial palsy. On the fortieth day there developed paralysis of tongue, vocal cords, and limbs, and on the sixth day after the onset of these symptoms he died from respiratory palsy. Extensive peripheral neuritis was the only nerve lesion."

In the following case, reported by Dr. Alfred Barrs, a period of three weeks elapsed between the healing of a wound and the onset of paralytic phenomena. The patient, a man aged 38, trapped the little finger of his left hand, and a few days later the finger was incised and some thin watery pus set free. There was well-marked inflammation of the lymphatic vessels of the forearm and of the left axillary lymphatic glands. In a fortnight the limb was quite well, with the exception of some stiffness of the hand and arm. When seen by Dr. Barrs, thirteen weeks from the date of the injury, the patient stated "that the stiffness of the left forearm and hand continued for three weeks after the injury, and then there appeared numbness in the fourth and fifth fingers of the left hand. Five weeks afterwards the feet became numb, and were the seat of pricking sensations, and two weeks later the right hand was similarly affected, so that at the end of seven weeks the hands and feet were in a similar condition so far as 'numbness and pricking' were concerned. At the same time there was marked motor weakness in all four limbs, and the legs were slightly stiff, as if from cramp. The feet were described as feeling loose at the ankle, and as scraping along the ground when walking. The gait was quite characteristic of weakness of the anterior tibial muscles." The knee-jerks were entirely absent. There was some wasting of the anterior tibial muscles, which gave a sluggish reaction to a strong faradic current. The weakness of the hands was extreme. "Careful testing failed to find any alteration of tactile sensation in any of the paralysed parts, but he stated that there has been some diminution of ordinary touch sense in the left hand." The special senses and the visceral functions were quite normal. Neither swallowing nor the voice had ever been affected; he had not had any sore throat, nor had there been any epidemic form of sore throat in the neighbourhood in which he lived. "There was no evidence of lead poisoning, or of any of the more generally recognised toxic conditions which produce peripheral palsies." After three months' treatment with tonics and gentle faradism he was practically well.

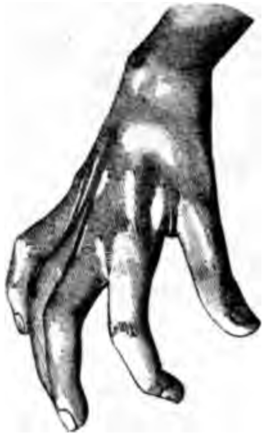
Dr. Ross, in his account of diphtheritic paralysis, has given a graphic description of the difficulties met with in attempting to find evidence of exposure to the poison of diphtheria; and similar difficulties may attend the diagnosis of septicæmia. Dr. Gowers says: "I have met with cases in which the symptoms of multiple neuritis, perfectly characteristic, came on without any discoverable cause and ran a course in most cases mild, and ending in recovery; in one or two others severe, and ending in death. Some cases of what is popularly termed 'blood poisoning' were probably of this nature."

Again, a paralysis following a wound may appear to be due to the virus of septicæmia when it really depends on that of diphtheria, the wound having been invaded by the organisms of that disease. It is indeed highly important in estimating the evidence in favour of the occurrence of paralysis after any of the acute specific diseases not to overlook the possibility of the chief disease being complicated by diphtheria.

The difficulty referred to is well illustrated by the clinical history of the following case, for the notes of which I am indebted to Dr. Thos. A. Goodfellow, Resident Medical Officer to the Infirmary.

Emily S. B., aged 15 years, was admitted as an in-patient to the Manchester Royal Infirmary, under the care of Dr. Ross, on October 13, 1888. The patient is a mantle-maker by trade, and with the exception of having had in childhood abscesses in the neck, the cicatrices of which are to be seen on the left side, she enjoyed good health until about three months ago, when she was obliged to cease work owing to a gathered finger. The finger was lanced and poulticed but it got worse under treatment—an ash-coloured slough formed over the wound, and healing was for a long time delayed. Soon afterwards an abscess formed in her back, and although it was not painful she felt very ill and took to her bed, where she remained for a month. Soon after getting up she began to complain of numbness and tingling in the fingers and toes, and a few days later she found herself unable to stand or walk without support, and objects slipped from her grasp. She was now obliged to return to bed, and the weakness gradually increased in degree and extent, so that in a few days she was unable to move her legs in bed, and the weakness, which was at first felt only in her hands, extended to the movements at the elbows and shoulders. About this time her friends told her she squinted, and she saw objects double, but she cannot remember whether the eye symptoms preceded or followed the weakness of the legs. She never suffered from sore throat, and had at no time any difficulty in swallowing, or ejection of fluids through the nose, alteration of voice, or disorder of articulation. She had also been free from pains or tremors in the limbs. Both her father and mother, and also a brother died from consumption. Two brothers and a sister are living and healthy.

Present Condition.—The patient lies in bed on her right side with the knees drawn up, the right knee being somewhat more flexed than the left, so that the left lies behind the right knee. She is a fair-haired pale-faced girl, of strumous appearance, and several cicatrices are seen in the left side of the neck below the ear, and behind the angle of the jaw. On a level with the tenth dorsal spine, there is an inflamed swelling about the size of a five-shilling piece, from which a thin sero-purulent fluid flows; but on exploration the cavity is found to be quite superficial, and to have no connection with diseased bone. The index finger of the right hand is deformed; the distal phalanx has a deep cicatrix running along the whole length of its palmar surface, the tip of the finger is tapering, and the last phalanx is flexed on the second, while the joint is stiff, so that the finger has the appearance of a hook. There is marked loss of motor power in all the extremities, and the muscles are manifestly wasted, the emaciation being most marked in the extensors of the forearms and anterior muscles of the legs. The backs of the hands are much grooved between the metacarpal bones, and the thenar and hypothenar eminences are flattened. When the forearms are held out horizontally in the prone position, the hands are somewhat dropped at the wrists, and although the patient can raise them momentarily she cannot maintain them in an extended position. The fingers are extended at the metacarpo-phalangeal and flexed at the interphalangeal joints, and she cannot approximate the tips of the thumbs and little fingers without



flexing the thumbs at the phalangeal joint. The lower limbs are maintained in flexed positions, but they can be extended without causing much pain by passive movements. When the patient lies on her back with feet extended, the feet are somewhat dropped at the ankles, and the big toes are flexed into the sole at both joints, while the little toes are hyper-extended at the metatarso-phalangeal, and flexed at the phalangeal joints. The patient can produce by voluntary effort slight flexion at the toes, a feeble extension of them, and of the feet at the ankles. She is unable to raise either foot from the bed, but can draw up the legs by flexion of the thighs, although this and all the other movements of the lower limbs are very feeble. The muscles of the back and abdomen as tested by the efforts of the patient to sit up in bed are feeble, but the diaphragm and intercostal muscles, as well as the muscles of the neck and head, appear to be unaffected. The patellar tendon reactions are absent on both sides, and there is no ankle clonus. The plantar reflexes are present, but very feeble and sluggish. The abdominal, epigastric, and palatine reflexes are active. The patient complains of numbness of her fingers and toes, and there is some loss of tactile sensibility in the tips of the fingers, but there are no other evident sensory disorders to objective examination. The sphincters of the bladder and rectum are unaffected. The sounds of the heart are somewhat lacking in clearness, and the pulse is feeble and compressible. The lungs and abdominal organs show no signs of disease on physical examination, and the urine is free from albumen and sugar. The patient was ordered to have a quinine mixture, cod liver oil, and massage.

November 16.—The patient has improved under treatment, and has regained a considerable degree of motor power in her limbs, and the muscles have become plumper. The small muscles of the hands, however, are still much wasted; the thenar and hypothenar eminences are flattened, and the intermetacarpal spaces are markedly grooved. The patient is now able to hold her hands extended at the wrists, but when she tries to separate her fingers they still assume the position already described, in which there is hyperextension at the metarcarpo-phalangeal, and flexion at the phalangeal joints.

The power of approximating the thumbs and little fingers is still much impaired, and the movements of the biceps, supinator longus, and triceps, as tested by opposing actions, are feeble.

December 4.—The patient has advanced greatly towards recovery since last report. She can now use her hands in sewing, and is able to stand and walk a little without assistance.

December 13.—The following electrical reactions were noted to-day:—

	Right.		Left.	
	KSC.	ASC.	KSC.	ASC.
Extensors of forearms ....cells Leclanché	20	30	20	25
Anterior extensors of legs ...... „ „	30	30	35	50

January 2, 1889.—During the last fortnight the patient has greatly improved in general health, as well as in motor power. The affected muscles are firmer and of greater volume than they were at the date of the last report, but the patellar-tendon reactions are still absent. The patient is unable to raise her right arm above the horizontal level, and during the effort the inferior angle of the scapula is drawn towards the vertebral column and stands out from the chest, while the superior external angle stands on a lower level than the superior internal one, the position assumed being due to paralysis of the serratus magnus of that side. The patient was seen a fortnight later, and she is reported as having completely recovered, but no note was taken of the patellar-tendon reactions or of the functions of the serratus magnus.

The question whether the paralysis in this case owed its origin to the poison of diphtheria or to that of septicæmia cannot be definitely settled. Certainly the absence of regurgitation of fluids through the nose, of alteration of the voice, or affection of the special senses, is much against diphtheria. At the same time the history of squint and double vision makes it not unlikely that the finger wound had become infected with the poison of diphtheria, and in that case the paralysis must be regarded as dependent on a diphtheritic neuritis.

ROTH.—*Corr. Bl. f. Schw. Aerzte.*, 1883, No. 13. Quoted by Gowers, Vol. I., p. 115.

BARRS (A. G.). "On the disturbances of the tactile sensory function of the skin in cases of peripheral neuritis."—*Intern. Journ. of the Med. Sciences*, 1889, p. 131.

DANA. "Septic peripheral neuritis due to pyelo-nephritis."—*Med. News*, 1889.

(*m*) SYPHILIS.

Syphilitic neuritis affects mainly the cranial nerves, and is then of the interstitial variety. Sometimes one of the spinal nerves is affected, as the circumflex (Hutchinson) or the sciatic, but a true multiple syphilitic neuritis is excessively rare. As Dr. Gowers remarks in his Lettsomian Lectures: "It is remarkable that the peripheral parts of the spinal nerves very seldom suffer. The discovery of the susceptibility of these nerves to various toxic influences has revealed no liability for them to be influenced by syphilis."

There are several published cases of acute ascending paralysis in syphilitic subjects. Dr. Ross has analysed eight of these, and if we accept his view, that acute ascending or Landry's paralysis is most commonly a form of peripheral neuritis, then the occurrence of syphilitic polyneuritis may be admitted. Parenchymatous neuritis occurs in locomotor ataxia of syphilitic origin, but this form will be discussed in Section IV., under the heading "Neuro-tabes Peripherica."

Leaving out of consideration these two groups of cases, we may ask if there is any other evidence to show that a multiple neuritis may be set up, if only occasionally, by the virus of syphilis. Perhaps the most satisfactory evidence in this direction is afforded by two cases reported in detail by Dr. Buzzard in the "Transactions of the Clinical Society" for 1874 and 1880. These cases are so important that we have no hesitation in giving the following brief account of them, taken from Dr. Buzzard's book on "Peripheral Neuritis":—

"W. H., a working man, aged 44, of previous good health, was brought to me at the hospital in January, 1873, in the following condition. He had double facial paralysis, total absence of power of voluntary contraction in the muscles of either leg, the grasp of both hands almost entirely lost, and partial paralysis of respiration and deglutition. There was incomplete paralysis of the right external rectus muscle and of the soft palate, especially on the left side. There was but little movement of the diaphragm, and the intercostal muscles were likewise acting so imperfectly that the patient could not lie down in bed. His sterno-mastoid and trapezii muscles acted freely. Cutaneous anæsthesia was more or less general throughout the trunk, extremities, and face—the tips of the fingers being especially numbed. The plantar reflex was absent in each foot. There was slight power of voluntarily contracting the muscles on the front of each thigh, but he was unable to contract in the least those on the front of either leg below the knee. A sense of numbness and weight was complained of in each leg, and occasionally a 'throbbing ran down the left thigh and calf.' For the first two or three weeks also he had suffered from 'pins and needles' in his legs. But at no time apparently had there been any actual pains in his extremities or involuntary muscular contraction. The power of the sphincter ani was normal, that of the bladder impaired to a slight extent. The muscles about the mouth showed the reaction of degeneration. In those of the arms the reaction to faradism was greatly diminished, whilst in those of the legs, below the knees, it was quite absent; in the left thigh it was greatly diminished. (The right lower extremity was lame and wasted from an old attack of infantile paralysis.) But in no part of the upper or lower extremities was there increased action to slow intermissions of the galvanic current. In the face, however, this was marked. The facial muscles reacted to interruptions of a current from six cells (Stöhrer). His attack had commenced one month previously with numbness in the finger-ends, followed on the same day by weakness in the legs, which increased next day and was then accompanied by numbness about the calves, thighs, and buttocks. The weakness increased day by day, and a week after the beginning of his illness he had the sensation of a tight band round his abdomen. A few days later he could use neither arms nor legs. The difficulty of swallowing was not observed till a fortnight after the onset. There had been no fever. There was nothing abnormal in the mental condition, nor in the heart, lungs, and kidneys. The patient was at once admitted and placed on a water-bed. For twenty-four hours his condition was one of imminent danger from the state of respiration. As there was a syphilitic history, he was treated with iodide of potassium, and later with mercury. He soon began to improve, and in six months was able to resume his employment. A few months later I showed him at the Clinical Society, entirely recovered.

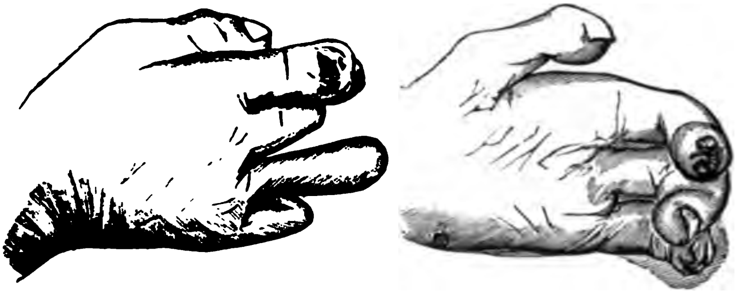
"On April 14th, 1879, T. O., aged 44, was admitted into hospital with paralysis of all four extremities and both sides of the face, together with inability to swallow solids. The respiration was mainly upper thoracic, and there was some loss of control over the bladder and sphincter ani. His grasp was feeble; he was unable to stand; as he lay in bed he could move one foot across the other, but could not lift either more than three or four inches off the bed. There was great muscular flaccidity. He did not know where his legs were. The knee phenomenon was absent on each side. Over the right side of the face there was complete loss of sensibility to touch and pain, with apparently increased (but at all events well-retained) sensibility to heat and cold. The anæsthesia was likewise observed, though to a less extent, over the left side of the face, and also, though here again in a less complete degree, on his forehead. He complained of great pain in the right half of the forehead, spreading towards the vertex. Below the middle of the forearm on each side there was almost entire loss of sensibility to touch and pain, whilst heat and cold were well recognised. Where the alteration in sensibility began there was what the patient described as a 'band-like feeling around the arm.' In the tips of his fingers there was a constant tingling sensation, and anything which he touched with them felt hot. In his lower extremities sensibility was also greatly modified. Below the middle of the thigh on each side neither a touch nor the prick of a pin could be recognised. Over the whole of both feet, as well as half-way up the legs, and on the posterior surface of the rest of each lower extremity, sensibility to heat and cold appeared intensified. Over the whole of the trunk, and in the extremities down to the boundaries described, cutaneous sensibility was normal. He had frequent pains like knife-stabs in the lower extremities, and the legs would twitch when they occurred. At other times the pains were of a dull heavy character. In this case facial paralysis on the left side seems to have been the first symptom which was observed by his friends a month before admission. The patient knew nothing of this, and felt quite well till a fortnight before I saw him. He then noticed 'pins and needles' in his hands and feet. Three days later he had diplopia, and his legs became weak. In five more days he could not walk, and there was difficulty in swallowing. In another three days he could not dress himself. On admission, and for a few days afterwards, reaction to induced currents was almost entirely absent in the muscles of the face, and also in the thenar eminence and interossei of each hand; it was lessened, though not to the same extent, in the muscles of the front and back of the forearms. There was very slight reaction to induced currents in all the muscles of the lower extremities. Under active mercurial treatment the patient entirely recovered in about six months. On August 10th the knee-phenomenon was found to have returned in the right leg, and three days later in the left. This patient was likewise shown at the Clinical Society."

Dr. Buzzard believes that these cases are examples of peripheral neuritis, and there appears to be a high degree of probability, amounting almost to a certainty in the first case, that the cause was syphilis, and not alcohol or any other toxic agent, for Dr. Buzzard says: "There is no doubt that the patient, W. H., was a temperate man. As regards T. O., he is described in my notes as having 'lived fast and drank fairly,' whatever that may mean. Both had had syphilis, and recovery in each case was absolutely complete under specific treatment."

The following is a brief abstract of a case reported by Taylor as one of syphilitic polyneuritis. It is chiefly remarkable (1) for the extensive affection of the cutaneous sensibility, along with preservation of muscular power and nutrition; and (2) for the "mutilations" of the fingers and toes.

A woman, aged 40, contracted syphilis about two years before coming to the Hospital for the first time, in 1882. In June, 1882, she had a typical syphilitic iritis, and copper-coloured stains over the body and legs; she also suffered from rheumatism, worse at night. Early in 1882 she noticed that the sensation on the backs of both hands was impaired, and when she had been in hospital a few weeks there was marked analgesia and anæsthesia over the backs of the fingers, hands, and wrists. She had pains in the eyes and dimness of vision, and the ophthalmoscope showed double neuro-retinitis. Under treatment the eyes quickly recovered, but the analgesia slowly extended up the arms, and was associated with severe aching and lancinating pains in the arms. In February, 1883, the patient began to complain of tenderness, pain, and swelling in the left heel, and soon after in the corresponding foot. The pain extended up to the knee, and was dull and apparently deep-seated. At this time a diffuse gummatous infiltration was observed on the cheeks and over the left ankle. There were also tender spots of periostitis over the cranium, together with severe headache at times. At the end of 1883 the anæsthesia and analgesia had extended up each arm to the shoulder, being complete on the extensor and partial on the flexor surfaces; an analgesic spot was also found on the dorsal aspect of the left shoulder. During all this time of increasing nervous disturbance the patient had complained of little, if any, impairment of muscular power. Towards the end of 1883, owing to the loss of sensation, she had burned, scratched, scalded, and in many ways injured and bruised her fingers.

At this time also she began to complain of numbness in the feet, and particularly in the toes. In January, 1884, there was analgesia of nearly the whole of the lower limbs, and during the whole of 1884 the analgesia persisted in both arms and legs. No thickening of the nerves of the brachial plexus could be detected. For months the patient suffered paroxysmally with severe headaches, which prevented sleep at night. Degenerative changes began in the fingers in the summer of 1884. These degenerative changes had their starting-point in indolent ulcers and bullæ, resulting from various traumatisms. First the skin and fibrous tissue disappeared, and then portions of bone came away in spiculæ, and in the form of detritus. When the degeneration was not very active and extensive, healing occurred, but in most instances painful deformities of toes and fingers were produced, which required surgical intervention to bring about sightly and tolerably serviceable stumps.



(After Taylor.)

At the present time, 1890—that is, ten years since the onset of the sensory disturbance in the hands—her condition is as follows: There is good power in both arms and legs, and no diminution of muscular sense nor ataxia. There is anasthesia over a great part of the face. Sensation is present in the trunk, though much blunted. In the upper limbs there is a total loss of sensation from the shoulders down, except a small fold at the elbow and a narrow strip on the inside of the arms, below the axillæ. There is also complete anæsthesia of the whole of the lower limbs. The patellar reflex is present, the plantar absent. The patient can walk fairly well, but owing to the mutilations of the hands, she has been unable to gain her living, and is capable of very little manual labour.

Dr. Taylor says that "several gentlemen of prominence" who saw the case were disposed to consider it to be one of leprosy, but he himself thinks that such a diagnosis is untenable, and points out that the analgesia appeared on the backs of the hands about eighteen months after syphilitic infection.

A case published by Ehrmann also affords proof that a neuritis of spinal nerves may develop in the active and earlier stages of syphilis. The case was that of a man thirty-eight years old, who entered the hospital on the 16th of December presenting a hard chancre and generalised secondary eruptions. In his urine a large quantity of albumen, cylindrical epithelium, red and white blood corpuscles, and epithelium from the pelvis of the kidney were found. Under the influence of hot baths and iodide of potassium internally he seemed better in about six weeks, and the albumen was no longer found in the urine. A little later on he became jaundiced, and on the 29th of April periostitis of the left tibia caused the resumption of the iodide. Then in a short time periostitis of the external malleolus of the left side, pain in the tendo Achillis, and in the gastrocnemii muscles, and swelling and pain over both cuboid bones, were complained of. Then it is noted that pains were felt in the first and second phalanges of the left ring finger, and a sensation of tingling on the ulnar side of the left forearm and in the ring and little fingers of the same side. Careful examination of the brachial plexus showed that the nerves were very sensitive to pressure in their whole length, notably, the ulnar nerve. Also the nerves on the left side were much more distinctly felt than those of the right and unaffected side. The thenar and hypothenar eminences and all the muscles supplied by the ulnar nerve were visibly atrophied. There was hyperalgesia in the regions of the internal cutaneous and ulnar nerves on the left side. Slight punctures with a needle produced small bullæ, surrounded by a red areola, which phenomenon Ehrmann regarded as evidence of vaso-motor disturbance. The electrical irritability of the ulnar and median nerves and the atrophied muscles was diminished. The knee jerks were present, and the tendon reflexes of the upper limbs were equally marked on both sides. A fair amount of improvement was produced by iodides, in doses of thirty grains daily, but the symptoms were still manifest in July.

In the above case there is proof of a neuritis involving several branches of the brachial plexus, but there are no indications of a more generalised disorder of the peripheral nerves. In addition to the cases of Taylor and Buzzard, one reported by Tuckwell may be quoted as probably an example of multiple neuritis from syphilis

It reads as follows:—

A woman, aged 30, in all probability syphilitic, was one day attacked with aching pains in the ankles and legs, and she noticed that her legs easily gave way when she tried to walk. She had also tingling sensations in the legs, and her feet were cold. A week later her arms gradually lost power, but without any numbness or unusual sensations in them. On examination there was paraplegia, with complete anæsthesia below the knees and partial anæsthesia in the thighs. The grasp of both hands was feeble, but the motor power of the arms was unaffected. The hands and parts of the arms were anæsthetic. The knee-jerks were abolished; the optic discs were blurred; the sphincters were normal. Nine days later anæsthesia was absolute over both hands and both forearms; the pains in the legs had increased; there was no rigidity or wasting of the muscles of the extremities. A fortnight later the patient had a convulsive attack, which began with twitching of the right hand and the right side of the face. Optic neuritis was now well marked. The movements of the hands presented considerable inco-ordination. Seven months later, having been treated in the meantime with large doses of potassium iodide and galvanised, she was much better. She could walk, and the upper limbs had completely recovered. The muscles of the left leg contracted to a weak faradic current, whereas formerly they did not respond to a strong one. There was still partial anæsthesia below the knees. The right optic disc was normal, the left one slightly blurred.

The spinal roots are probably more commonly affected by syphilis than the peripheral nerves, and, as a rule, in association with lesions of the membranes or vertebræ. But meningeal symptoms may be slight or absent, and then a radicular neuritis may present symptoms closely resembling a peripheral neuritis. In the following case, published by Kahler, the spinal roots were affected, while the spinal membranes appear to have entirely escaped.

A man, aged 29, three months after contracting syphilis, had left hemiplegia of the ordinary type. Articulation was much disturbed, but there was no aphasia. Inunction with mercury produced no improvement, and the following phenomena gradually developed. There was first complete left and then complete right facial paralysis, both of the peripheral type; then paralysis of both thirds; vaso-motor disturbances; atrophy of the muscles of the paralysed extremities; finally, attacks of severe neuralgia in the region of the occipital and intercostal nerves. Death from lung disease ensued 5½ months after the onset of hemiplegia. The autopsy revealed slight syphilitic changes in the cerebral membranes and basal vessels; a softened focus, the size of a hazel nut, in the middle of the right half of the pons; and neuritis of the roots of various cerebral and spinal nerves. The affection of the spinal roots was quite independent of any lesion of the membranes.

Finally we may mention a case reported by Dr. Barrs, which, though not strictly belonging to our present subject—"multiple neuritis"—is a marked example of localised neuritis, and furnishes us with a suggestive link between syphilis and Raynaud's disease.

A woman, aged 34, came under Dr. Barrs's observation in September, 1890. "There have been syphilitic manifestations of one kind or another, fairly continuously during the last four years. At first there were some pains, mainly in the head, accompanied later on by falling off of the hair, and a sore throat of three weeks duration." These were soon followed by ptosis and external squint of the eye, which lasted twelve months. About the same time the fourth and fifth fingers of the left hand began to "go numb"; they would turn blue and dead on exposure, but were quite right when kept warm. Twelve months later the fingers assumed their present position. About two years ago lumps began to be felt in the left arm, mainly about the elbow. Recently the right popliteal space became very painful, and the condition was much relieved by iodide of potassium.

Present Condition.—The patient is a pallid decrepid woman of low intelligence. The remains of the right third nerve paralysis are still present. The left arm is held in a semiflexed position, and cannot either by force or will be completely extended. Flexion and extension at the wrist joint are limited. The little finger is completely and firmly flexed in all its joints; the ring finger is flexed at its first and second joints, but not so much at its third. The position is precisely that of Dupuytren's contraction, but there is no affection of the skin or fascia of the palm. The two fingers are extremely cold, and the skin over them is hard, thin, and glazed; the little finger is exquisitely tender to touch, and the fourth finger moderately tender. There is a livid somewhat raised patch over the second joint of the fifth. The skin covering the hypothenar eminence is much thinner and more deeply coloured than the rest of the palm. There is no anæsthesia and no muscular atrophy. The muscles of the upper arm are hard and shortened.

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Buzzard (T.). "A case of rapid and almost universal paralysis involving the four extremities, both sides of face, respiration, deglutition; syphilitic history; recovery."—*Transactions. Clin. Soc. Lond.*, 1880, Vol. XIII., p. 180.

Tuckwell. "Paralysis, probably syphilitic, affecting in rapid succession both legs and arms; impaired vision; optic neuritis; gradual recovery under large doses of iodide of potassium."—*Lancet*, 1882, Vol. I., p. 62.

Ehrmann. "Ein Fall von halbseitiger Neuritis spinaler Aeste bei recenter Lues."—*Wiener mediz. Blätter*, 1886, Nos. 46 and 47.

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FORDYCE (J. A.). "Peripheral neuritis of syphilitic origin."—*Jour. Cutan. and Genito. Urin. Diseases*, New York, 1891, Vol. IX., p. 174.

BARBE. "Paralysie des membres supérieurs chez une petite fille atteinte de syphilis héréditaire."—*Bull. Soc. franc de Dermat. et Syph.*, 1891, Vol. II., p. 85.

(*n*) PNEUMONIA.

It is to my colleague Dr. Leech that we owe the most important contribution to the subject of peripheral neuritis following pneumonia. In a paper published in the *Medical Chronicle* he gives a complete *résumé* of the literature relating to paralysis after pneumonia, together with an exhaustive account of the clinical history of a patient who had been under his care at the Manchester Royal Infirmary. In the case referred to the attack of pneumonia, except that it was infectious in origin, ran an ordinary course and terminated by crisis on the seventh day. During convalescence, however, the evening temperature for the most part was slightly above the normal, and the physical signs of lung change lasted an unusually long time after crisis had taken place.

About a fortnight after the first symptoms of pneumonia had shown themselves, and four or five days after crisis had occurred, loss of motor power had become manifest in the lower extremities, but it had come on so gradually that no actual date could be fixed for its commencement; and, furthermore, indications of sensory changes were noted antecedent to distinct loss of power. The patient had spontaneously complained of a feeling of slight numbness of the hands, and a "queer" feeling of the skin of the abdomen and feet. His condition on the eighth day of convalescence is thus recorded by Dr. Hopkinson: "There is no spasm or tremor anywhere, the movements of the fingers appear normal, the thumb can be brought into apposition with the little finger, yet he picks up a pin rather clumsily, and the grasp of the right hand is feeble; the left hand was amputated after an accident twelve years ago. There is a marked loss of power in both legs, the left being weaker than the right. He is unable to stand, and when lying down cannot flex the left thigh on the trunk or adduct the limb. The first phalanges of the toes are hyperextended, the muscles of the limb flabby, but not atrophied. Though flexion of the leg on the thigh is easily performed, extension of the leg is slow and difficult.

"On the right side flexion and adduction of the thigh are not so much affected, but otherwise the condition is the same as on the left side. The patient can only sit up in bed with difficulty, and the muscular movements generally seem enfeebled. No definite loss of power can be detected in the ocular and lingual muscles. The knee-jerk is absent on both sides. The plantar reflex is normal; the cremasteric, abdominal, and epigastric are not easily excited. There is distinct sensory disturbbance; common sensibility is rather blunted, though not absent anywhere; he has cramp-like pains in his fingers, with slight numbness, and he says he has at times a feeling there like 'pins and needles.' When the muscles of the calf are grasped firmly, he complains of pain, but he has no pain or tenderness over the spine. The pupils react to light and accommodation, and the fundus oculi is normal."

Three days later slight nystagmus and imperfection in the movements of the upper eyelid were observed. There was hyperæsthesia of the arm muscles and marked loss of power in the abdominal muscles. The power of flexing the first phalanx of the thumb was lost, and the patient was unable to raise himself in bed without assistance. The bladder and rectum were normal. No inco-ordination of movement or want of muscular sense was at any time detected. The contractions caused by the induced current applied to the muscles of the lower limbs were normal; but the influence of the galvanic current was slightly modified. Shortly afterwards he began to recover slowly, but it was three months before his walking power was completely restored.

In his discussion of the etiology of Landry's paralysis, Dr. Ross is inclined to attribute cases of paralysis which have been described as occurring after pneumonia, to a diphtheritic poison, but Dr. Leech was unable after a careful investigation to get any evidence that his patient had presented symptoms of diphtheria, nor was diphtheria prevalent in the neighbourhood where the patient lived. And as to the possibility of alcoholic poisoning, it is stated that "there is no reason to believe that he had usually taken more than a glass or two of beer daily." Hence Dr. Leech believes that the paralysis in his case, and in some other published cases, was "due to a specific poison produced in some way we know not of during the pneumonic process," and he suggests that "it may eventually be found that cases of pneumonia, in which there is evidence of infection, may be more frequently followed by affections of the nervous system than those in which such evidence is wanting."

GUBLER. *Loc. cit.*

HUXAM. "Essay on fevers." 1775.

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Boulloche. "Des paralysies pneumoniques."—Paris: Steinheil. 1892.

(*o*) Tubercle.

Eisenlohr and Joffroy were the first to prove the existence of peripheral neuritis in tuberculosis.

Joffroy's case, already referred to by Dr. Ross (p. 9), was that of a woman, aged 33, who suffered from pulmonary tuberculosis and marked feebleness of the lower limbs. When admitted to the hospital she could walk with assistance, but eight days later there was almost complete paraplegia. There were no contractions, and sensibility to pain, touch, and temperature was quite normal. The knowledge of the position of her legs, however, was quite lost. The plantar reflex was diminished. The upper limbs were normal, also the bladder and rectum. A fortnight later, feebleness of the upper limbs developed, the extensors of the hand being especially paralysed. There was also some degree of inco-ordination in the movements of the arms, and the sense of position was lost; thus the patient in trying with her eyes shut to touch the tip of her nose with the end of her forefinger, touched instead her cheek, her eye, or her forehead. This was all the more remarkable, because previously it had been ascertained that she could perform such a movement with great precision. At this time there was appreciable atrophy of the muscles of the lower extremities. A week later the thenar and hypothenar eminences, the muscles of the forearm and the deltoids were markedly atrophied. The atrophy increased, and the affected muscles did not respond to a strong faradic current. The patient died after being in hospital one month. The cord and its membranes, as well as the spinal roots, were found to be perfectly healthy, but the peripheral nerves of the limbs showed marked changes. In all the preparations examined there were a number of fibres in which the medullary sheath was broken up and disseminated with fat granules.

The case is an interesting example of atrophic paralysis without loss of the cutaneous sensibility from peripheral neuritis. It is of especial interest, too, on account of the loss of the muscular sense, which symptom, as Joffroy points out, was observed in a case where the integrity of the cord and of the posterior roots was anatomically proved.

Since the publication of the above cases abundant testimony has been brought forward to show that the peripheral nerves taken from tubercular subjects frequently exhibit changes typical of a genuine parenchymatous neuritis, while a careful study of the recorded cases gives strong support to the conclusions formulated by Pitres and Vaillard, namely: That the neuritis is developed independently, and is not related to any pre-existing lesion of the brain or cord. That it may attack sensory, motor, or mixed nerves, and may also affect the cranial, the pneumogastric, or the phrenic nerves. That the symptoms of neuritis in tuberculosis are very complex and variable—in some cases muscular atrophy, localised or generalised, is predominant—while in other cases sensory phenomena, as hyperæsthesia, anæsthesia, and neuralgia are chiefly present.

Frequently peripheral neuritis is discovered after death when no symptoms of such a lesion were observed during life. The existence of this "latent neuritis" has been confirmed by the investigations of Jappa, who made a histological examination of a large number of nerves taken from fifteen cases of chronic pneumonia and pulmonary tuberculosis. In all the cases nerve fibres were found in a state of parenchymatous neuritis, and as a rule the peripheral branches were more affected than the trunks of the nerves, and those going to the lower more than those going to the upper limbs.

As to the pathology of tubercular neuritis Leyden believes that it properly belongs to the cachectic variety, being dependent on lowered vitality rather than on any specific poison. While this explanation may hold good for many cases where muscular atrophy is the leading feature, it appears to be scarcely adequate for cases of occasional occurrence, in which motor and sensory symptoms are present, characteristic of a true multiple neuritis. Then if we can exclude the influence of alcohol and other poisons, it appears justifiable to conclude that the neuritis results from the action of a specific poison generated by tubercle bacilli.

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(*p*) Malaria.

Dr. Gowers refers to several cases "in which persons living in districts where remittent fever is endemic, have suffered from weakness in the legs, chiefly in the muscles of the foot, and far greater in the anterior tibial group of muscles—the flexors of the ankle and extensors of the toes—than in any others. In most cases the paralysis of these muscles was absolute, and they presented the reaction of degeneration." "In all, the sphincters were unaffected. The symptoms steadily improved, quinine being given and the muscles stimulated by voltaism." "The disorder must be regarded as a peripheral neuritis, involving chiefly the motor nerves and confined to the legs, at least in cases of moderate severity." Dr. Buzzard also mentions a case of atrophic paralysis involving the flexors of the left foot and associated with some anæsthesia of the corresponding surface; these symptoms came on two years after a severe attack of dengue, but the patient had also suffered from ague-like attacks more recently.

There can be little doubt that these are examples of localised neuritis, which in all probability was excited either by specific organisms or by some poison produced by them. But there are a number of other cases on record of "malarial paralysis," the pathology of which is by no means clear. We allude to cases in which motor paralysis comes on very acutely, either during a fit of ague or quickly after it, is often almost universal in its distribution, and then may pass away, sometimes in a few days, sometimes in a few months. Such an attack may be the only one, or the paralysis may occur intermittently or periodically. It may be associated with anaesthesia, with impairment of the speech, special senses, or mental functions, but is unaccompanied, as a rule, with any decided disorder of the bladder or rectum. This group of malarial palsies is well illustrated by the following records:—

Hartwig relates the case of a robust man, aged 23, who in the autumn of 1873, five years after an attack of tertian ague, began to suffer from intermittent total paralysis of the muscles of legs, trunk, arms, and neck, with impairment of speech, deglutition, and respiration, beginning at night, unaccompanied by fever or sensory disturbances, but attended with copious sweating, and recurring every twenty-four hours. In hospital the condition became aggravated, the attacks lasting 40 hours, the intermissions only 6—8 hours; full doses of quinine apparently warded off the attacks. In March, 1874, the patient had a fresh attack, during which the reflexes were abolished and the excitability of the paralysed muscles almost completely so. Although quinine at first seemed to prevent the attacks, they soon returned, and presenting usually a tertian type, persisted till his discharge about eight months afterwards. In the intervals considerable paresis of the muscles with contracture and diminished electrical excitability was present; on only one occasion was there a rigor and rise of temperature to 40° C. The attacks were apparently sometimes warded off by movement, and the paralysis, which commenced in the lower extremities, at the periphery, extended to the trunk, afterwards to the upper extremities, spreading from the trunk to the periphery. Recovery began at the periphery, first in the arms, afterwards in the legs.

Macario reports the case of a woman who, while suffering from an attack of intermittent fever, complained one day of a tingling in the feet, which successively invaded the legs, thighs, trunk, and upper extremities, and was quickly followed by a general sensory and motor paralysis. The tongue was so much affected that the patient's speech was almost unintelligible, and deglutition was difficult. Three hours after the invasion of the first symptoms the fever subsided, and the nervous symptoms which were apparently so grave rapidly disappeared. On each of the following two days the patient had a similar attack of fever, accompanied by paralysis, and on the fourth she had an attack which lasted eight hours, but under treatment with large doses of quinine, both the febrile and the paralytic phenomena finally disappeared.

Dr. Gibney has recorded two cases of paralysis, probably malarial in origin, occurring in children of six and seven years old. In the first case there were four separate attacks of paralysis affecting the lower limbs each time, and once the upper limbs also. They were preceded by fever, and malaria was suspected in the house where the child lived. Electrical reactions of degeneration and wasting of some muscles were observed. Recovery took place within times varying from a week to six months, being aided by removal from home and the administration of quinine.

In the second case there were three attacks, each preceded by fever, during which pains in the legs had been a prominent symptom. Two weeks after the last attack the child had convulsions, and finally died of exhaustion. No post mortem. In neither case was the spleen enlarged.

Dr. Gibney suggests that the pathology of the disease may consist in a congestion and œdema of the cord, analogous to that of the spleen.

Westphal and Consot have recorded some very remarkable cases of periodic paralysis, which had no certain relation to any previous illness.

Westphal's case was that of a boy who, four years previously, had severe scarlet fever, followed by another illness, probably Bright's disease, but there was no evidence of malarial infection. The paralysis came on in the afternoon, by midnight was complete, and disappeared during the following day. All four extremities were affected, the loss of power was absolute, and the nerves and muscles completely lost their electrical reactions during the periods of paralysis. There was no plantar reflex but the knee jerk, and cremasteric and abdominal reflexes were normal. Sensation and consciousness were unimpaired. In one of the later attacks there was total loss of the knee jerks, but recovery of voluntary movement and faradic excitability of the muscles were re-established in seven years. The attacks recurred for several months, but finally ceased.

Suckling mentions the case of a man who was admitted into the Queen's Hospital, Birmingham, suffering from paralysis in both his legs. He had had a fit of ague a fortnight previously, and during the last ten years a great many attacks. When the present attack came on he felt as if he had received a blow of a fist on the back of his neck; he felt giddy, lost power in his legs, and fell. He says that sensation was lost in his legs. Power gradually returned to his legs during the fortnight he was in the hospital. During the next year he had several attacks of ague, the seizures occurring daily, and being marked by a cold and a sweating stage. The day after the last attack he had a pain round the middle of his body, sudden giddiness, loss of power in the legs, partial loss of consciousness for a few minutes, and passed his urine involuntarily; he also lost his speech for an hour, and suffered from headache. A fortnight later he was again admitted into the hospital, when it was found that he had lost all power and feeling in the legs, and his speech was thick and slurred. On the next day sensation was perfect, and he stated that it returned during the night with a feeling of "pins and needles." There was still complete paraplegia. The plantar and cremasteric reflexes were absent; the knee-jerks were normal. There was no pain or tenderness in the spine or lower extremities. There was slight impairment of speech, but the memory and intelligence were unaffected. Involuntary micturition was still present, but later in the day he recovered power.

The nerves and muscles of the lower limbs responded normally to the faradic current. On the following day the legs regained power, and at night the patient could walk by himself, and could pass his urine naturally. The cremasteric reflex had returned, but the plantar was still absent. Two days later the patient was quite well, the plantar reflex was present on both sides, but the knee-jerks were slightly exaggerated.

A man, aged 21, under the care of Da Costa, was admitted to hospital on September 2nd. His illness began seven days before, with intense occipital headache and a violent chill followed by fever. The fever continued for three days; then another chill occurred and he became delirious. There was a third chill the day before admission. On admission he was suffering from a severe attack of remittent fever. Under large doses of quinine and phenacetin the fever subsided in two days. He sweated profusely and complained of great thirst. On September 8th, he had another chill and a temperature of 105°, and the same happened on the 16th. During the profuse sweating which followed the last outbreak he became wildly delirious, though the temperature had fallen to 99°, and he remained delirious all night, but was quite himself in the morning. By the 20th of the month the patient was sitting up; he looked pale and complained of weakness in his legs. An iron mixture was added to his treatment. On the 23rd cramps were noticed about the knees and he had vertigo. Quinine, four grains every four hours, was ordered. There was no return of the chills, but by the 24th the weakness of the lower extremities had culminated in complete paralysis; motion and sensation were alike lost; the arms were not affected. Soon, however, they, too, became weak. Iodide of potassium in large doses was now given instead of quinine.

During the next three weeks there was not much change in his condition. The arms showed slight weakness and the grip of the right hand was weaker than that of the left. Marked rhythmical trembling was observed and the fingers were extended. The right leg was completely, and the left leg almost completely, paralysed. Both knee-jerks were exaggerated, particularly the right one. There was no ankle clonus. The muscles were firm and well nourished. Sensation in the legs was completely abolished. The rectum and bladder were unaffected, and the temperature was normal.

The palsy now improved slightly, and he went around the ward on crutches. Arsenic was substituted for the iodide. On the 24th of

October he had a slight chill, followed by fever. There was no marked change in his condition till November 5th, except that he could now raise the right leg from the floor. He had some difficulty in emptying the bladder, and complained of a gnawing pain in the back in the mid dorsal region, which was greatly relieved by a blister. There was decided anæsthesia both in the legs and arms, together with formication in the legs; there was also a zone of anæsthesia encircling the chest about two inches below the nipple. He had almost complete loss of taste. Disorder of co-ordination was decided, and his gait was that of an ataxic, he could not stand with his eyes closed or walk in the dark. The muscular response to the faradic current was good. The pupils reacted normally to light and to accommodation.

On the 5th of November a group of head symptoms became manifest, and during the next nine days the following observations were made: Severe occipital headache, marked impairment of hearing; bi-temporal hemianopsia, great defect of memory, poor appetite, urine normal. Outbreaks of hallucinations and of maniacal delirium also occurred, and necessitated his being confined to bed with straps.

By the 14th, although there was still some evening fever, the patient's condition had greatly improved. Dr. Harlan, who examined his eyes, found that vision was much diminished and existed only in small nasal fields, which were sharply defined and perfectly symmetrical; the ophthalmoscopic appearances were quite normal.

On the 17th he had a maniacal attack; on the 18th he suddenly became almost quite blind, he could not distinguish objects, but could recognise some colours and see light on the nasal fields, while perception on the temporal sides was entirely lost. Twenty-four grains of quinine were now given daily, and he soon began to improve, and in a month left the hospital "fully convalescent and rejoicing in the free use of his limbs," When the symptoms were severe Dr. Leidy found in the blood elements which Laveran has described as characteristic of malarial poison, namely, pigmented bodies in the interior of red corpuscles, hyaline bodies, and pigmented crescentic bodies. During convalescence these bodies could not be discovered, although frequent examinations of the blood were made.

Da Costa believes that malarial paralysis occurs "rather in the irregular than in the typical cases of the disease, and that it is the outcome of malarial cachexia quite as often as, or oftener than, of acute outbreaks of malaria."

He distinguishes three varieties of paralysis:—"(1) The commonest form of general paralysis or paraplegia with irregular symptoms, of which the case related is a marked illustration. (2) The form in which the periodicity is striking, and which is more apt to show itself as a hemiplegia. (3) The rarest form, in which actual organic disease is produced by the malarial poison, and in which periodicity and variability are not prominent, the case running much the course of ordinary paralysis when produced by its usual causes."

Da Costa, therefore, holds the common belief, that malarial paralysis is strictly periodical, to be erroneous.

As regards the cause of malarial palsies, he suggests "that the malarial germs carried about in the blood may have a direct action on special nerve centres; not a mechanical one, but one such as several vegetable poisons possess, or as the poison of diphtheria exerts."

It would be unprofitable to speculate on the nature of these curious cases of paralysis. Some of them appear to be closely related to the cases of universal paralysis described by Dr. Buzzard as occurring in connection with syphilis (see page 297), and it may be that such forms of paralysis, temporary in duration, widespread in distribution, sometimes periodic in character, and occurring in connection with malarial taint, or sometimes independently of any previous disease, are better explained by acute poisoning of the peripheral than of the central parts of the nervous system.

A side light is thrown on to the pathology of malarial palsies and a partial support given to the hypothesis of their peripheral origin by a consideration of a paper from the pen of Macnamara. In this paper he narrates four cases in which dimness of vision, sometimes proceeding to complete blindness, quickly followed a fit of ague, and was found to depend on well-marked neuro-retinitis. The optic discs were indistinct, or completely obscured by effusion which extended into the retina; the retinal veins were congested and tortuous. Under treatment with large doses of quinine, arsenic, or strychnine vision gradually returned, and within three months was usually quite normal. Macnamara points out that there was an entire absence of symptoms indicating either meningeal or cerebral disease; that there was no evidence of syphilis, rheumatism, or kidney mischief, and that, although the patients had enlargement of the spleen, they were not suffering from marked anæmia. Recovery of sight, however, he states, is not the ordinary course of malarial neuro-retinitis, "for if the fever continues, atrophy of the optic papilla and total blindness too often occur."

In his fourth case there was also paralysis of the muscles supplied by the left ulnar nerve, and, to some extent, of the soft palate.

Macnamara thinks it probable that the inflamed state of the optic papilla is due to something of the nature of a microbe, which becomes planted in the affected tissues, and growing there produces ptomaines, which, in their turn, cause irritation of the tissues, engorgement of the vessels, and transudation of serum and leucocytes into the retina and optic papilla, and he suggests that analogous changes of other nerves may explain symptoms referable to them.

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(q) BERIBERI OR KAK-KÉ.

The chief symptoms of this disease depend on a multiple peripheral neuritis, which usually runs a chronic course, but like other forms of peripheral neuritis, may occasionally develop with great rapidity and severity.

The earliest symptoms, according to Pekelharing and Winkler, consist in a slight degree of the reaction of degeneration in the peroneal nerves and the flexors of the ankles. About the same time or a little later certain subjective sensations are experienced, such as heaviness and numbness in the legs, tingling, creeping, or burning sensations, together with palpitation and undue excitability of the heart. Then a progressive paralysis and atrophy set in; the flexors of the ankle are first attacked, and now give the complete reaction of degeneration to electricity; subsequently the calf muscles, the extensors of the knee, and other muscles of the lower limbs become affected. The trunk muscles may be involved, and in severe cases the muscles of the upper extremities, the extensors of the wrist, being, as a rule, the first to be affected.

The face often suffers and sometimes the muscles of the eyeball and larynx. The atrophic paralysis is associated with marked sensory disturbance. Tactile sensibility and the muscular sense are (according to Scheube) prominently affected; sensibility to pain is usually preserved, but the temperature sense and the cutaneous sensitiveness to faradism are often diminished.

Œdema is one of the characteristic features of the disease. In some cases it is very slight and limited to the legs and ankles, but in other cases it spreads widely, until at length it becomes general anasarca, and involves also the serous cavities, especially the pericardium.

Patients suffering from beriberi are anæmic, and complain of palpitation and dyspnœa, symptoms largely due to cardiac weakness and dilatation of the right side of the heart.

Death commonly results from cardiac failure, or sometimes from paralysis of the diaphragm and intercostal muscles.

The peculiar features of beriberi are the association of dropsy and signs of cardiac failure with symptoms of a multiple neuritis. Dropsy and dilatation of the heart are not uncommon in alcoholic paralysis, but they are less constant in occurrence than in beriberi. The former condition is also distinguished by the presence of severe pains and muscular hyperæsthesia, and by the fact that the arms are often paralysed as soon as the legs.

Beriberi, however, presents many variations in its course and in the combination of its symptoms. These have been classified by Scheube as follows:—

(1) A slight form with moderate weakness of the legs, œdema and cardiac palpitations, recovery taking place after several weeks or months.

(2) An atrophic form characterised by weakness of the legs, going on to complete paralysis and atrophy. Then paralysis of the arms comes on, and occasionally spreads to the face, tongue, palate, and larynx. Sensory disturbances are usually marked, but anæsthesia is rarely complete. Œdema is slight or absent. This form results in recovery after a course of one or two years, or in death through some secondary complication.

(3) The dropsical or dropsical atrophic form. Here general anasarca is combined with symptoms of multiple neuritis.

(4) The acute pernicious form. There is a rapid development of paralysis and œdema, while palpitation, dyspnœa, cyanosis, and weakness of the pulse, are also prominent symptoms, and lead to a fatal issue usually in a few weeks.

Pathology.—The investigations of many able observers furnish abundant evidence that the most constant changes in beriberi are : (1) A degenerative neuritis which affects not only the nerves of the extremities, but also in some cases those of the trunk, heart, and larynx ; (2) an excess of fluid in the cellular tissue and in the pericardium and other serous cavities. Characteristic organisms, in the form of little rods and diplococci, are almost invariably found in the blood, and cultures from the blood, when injected into rabbits, have produced a multiple nerve degeneration. But the organisms have not been found in special association with the diseased nerves, and it is probable that a chemical poison is produced during the growth of the organisms which attacks the peripheral nerves, and is peculiar in exhibiting a special affinity for the cardiac branches of the vagus.

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(r) ANÆSTHETIC LEPROSY.

The nervous symptoms of leprosy consist of muscular wasting and anæsthesia, in association with patches of pigmentation and pallor of the skin and mutilations of the fingers and toes. Distinct enlargements also may frequently be felt along the course of various nerves. The paralytic phenomena generally first appear in the distal portions of the extremities; in the upper limb the territory of the ulnar nerve is usually first and most severely affected; in the lower limb that on the outer side of the leg and foot. One of the peculiar features of the disease is the irregularity in the distribution of the symptoms, in consequence of the unequal way in which the fibres of the peripheral nerves are diseased. Thus anæsthesia may not correspond in area to the distribution of any particular nerve or nerves, or the knee-jerk may be obtained when the extensors of the knee are partially paralysed.

This irregularity in the distribution of the symptoms sometimes makes a case of anæsthetic leprosy present a close resemblance to one of syringo-myelia.

The neuritis of leprosy, upon which most of the nervous symptoms depend, is peculiar in being typically adventitial. The morbid process probably begins in the sheath of the nerve and in the sheaths of the fasciculi. These parts become thickened, and ultimately the development and growth of connective tissue in the interior of the fasciculi, and even between the nerve fibres, lead to slow wasting of the essential nerve elements.

The characteristic bacilli of leprosy are found in great numbers, at least in recent cases, in the new adventitial tissue, but as this grows and shrinks, the bacilli gradually become destroyed.

Recently Carl Looft examined two cases of anæsthetic leprosy, and found distinct degeneration of the posterior columns of the spinal cord. In both cases there was marked atrophy of the posterior roots, together with a fibrous degeneration of the ganglia. The peripheral nerves showed a chronic neuritis. The anterior roots, and the cells of the anterior and posterior horns presented no obvious changes. Looft believes that the primary change was in the posterior roots and ganglia, the cord being secondarily affected. The paralysis evidently depended on disease of the peripheral nerves, for both anterior roots and horns were quite normal.

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II.—TOXIC MULTIPLE NEURITIS. (3) THE NEURITIS CAUSED BY METALLIC POISONS.

(*a*) LEAD PARALYSIS.

The paralysis that results from lead poisoning is usually bilateral and symmetrical; most commonly the extensor muscles of the wrist and fingers are attacked, but sometimes other groups of muscles become involved, while occasionally the paralysis is generalised and attacks nearly every muscle of the body.

Paralysis may be the first and only manifestation of saturnine poisoning, but generally it occurs in persons who have been subject to attacks of constipation, colic, headache or vomiting, and who present a peculiar form of anæmia, as well as a blueish black line at the junction of teeth and gums.

Frequently too, local sensory phenomena precede and usher in the paralysis, just as in other forms of toxic paralysis. Thus the patient may complain of numbness and tingling in the extremities, of cramp in the calves, of dull aching pains in the joints, or of sharp pains along the course of the nerves. These sensory prodromata are usually slight in degree; they are often entirely absent, and certainly never present the constancy and intensity of those met with in connection with alcoholic paralysis. Sometimes paralysis comes on acutely, as during an attack of colic; at other times its development is very slow, increasing difficulties in the execution of certain movements being gradually noticed by the patient; but most commonly it develops in a subacute manner.

The paralysed muscles undergo rapid atrophy, and exhibit the reaction of degeneration to electricity. In the common type of paralysis affecting the muscles on the back of the forearm the faradic contractility, while lost in some of the paralysed muscles, is only slightly diminished in others, and it may be found that the area of paralysis is greater than that of altered electrical reactions. But the reverse holds, according to Remak, in some of the rarer types of paralysis; that is, while one or two muscles are paralysed, other muscles which act functionally with them, although not manifestly weak, exhibit altered electrical reactions.

Fibrillary contractions occasionally accompany the muscular atrophy. Tremor, however, is more common. It is fine in quality, and is increased by voluntary movement, and especially by fatigue. Usually limited to the upper extremities, it may become general in aggravated cases. Thus Dr. Suckling showed a man at a meeting of the Midland Medical Society who had worked in lead for many years. He had tremor in all four extremities, and could scarcely walk. The head and trunk were affected, and the muscles of the face, lips, and tongue were the seat of well-marked tremors, very similar to those seen in cases of general paralysis. The tremor ceased when the parts were at rest.

Alterations in the cutaneous sensibility are often absent in cases of lead paralysis; but when the muscles supplied by the external popliteal are paralysed, then a band of anæsthesia may frequently be detected on the outer aspect of the leg; but in the common wrist drop type of paralysis loss of tactile sensibility is rare. Sometimes an ill-defined zone of partial anæsthesia may be discovered on the posterior aspect of the forearm, hand, or thumb, or, as I have observed, in the territory of the circumflex nerve when the deltoid is paralysed. A diminution of sensibility to the faradic current, however, is constantly present; much stronger currents can be borne over paralysed than over healthy parts, and the same difference holds with regard to the nerve trunks, a patient bearing the application of a strong current to the musculo-spiral better than to the median or ulnar nerves. The knee-jerks present no constant relation to the distribution of the paralysis; they are frequently normal, and may be exaggerated or lost when paralysis is limited to the upper limbs.

The different localisations of lead paralysis may now be briefly described under the following headings:—

(1) *The Common or Wrist-drop Type.*—This is by far the commonest variety of lead paralysis. The common extensor of the fingers is usually the first muscle to be affected. Its weakness is shown by inability to extend the first phalanges of the two middle fingers; when these are passively straightened the patient can readily extend the distal phalanges by means of the unaffected interossei and lumbricales. Then the special extensors of the index and little fingers, the extensor secundi and primi internodii pollicis are successively involved; soon after the extensors of the wrist become weak, and ultimately the characteristic attitude of lead paralysis is assumed.

The hand, semipronated, is dropped and forms a right angle with the forearm, the fingers are slightly flexed and the thumb is drawn inwards, towards the palm. The hand is usually inclined towards the ulnar side, and power to extend it becomes completely lost.

The flexors of the fingers are unaffected, yet their action is much interfered with by the weakness of the extensors; when, however, the hand is raised, the flexors can act with vigour.

The extensor ossis metacarpi or abductor pollicis is usually spared, or if affected it is not till some time after the other extensor muscles have been paralysed. The supinator longus also escapes, but with those two exceptions all the muscles innervated by the musculo-spiral nerve are attacked in this common variety of lead paralysis.

(2) *The Upper Arm Type.*—In this form there is paralysis of the deltoid, biceps, brachialis anticus and supinator longus; sometimes the supra- and infra-spinatus are also involved, while in rare cases paralysis of the pectoralis major is superadded. As a rule, paralysis of this group of muscles succeeds that of the extensor muscles of the forearm, but it may be primary, and then the deltoid is usually the first muscle to be attacked. It may, indeed, be the only muscle of the group to be paralysed; this was the case in a patient under the care of Dr. Buzzard. In this patient the left deltoid was almost completely paralysed, but only slightly wasted, and it is remarkable that the electrical reactions of the deltoid and of other muscles tested were quite normal. Generally, however, in this upper arm type, changes in the electrical reactions and atrophy of the muscles are much less marked than in the ordinary type of paralysis.

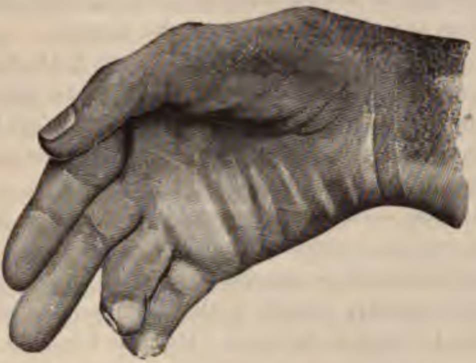
Sometimes paralysis and atrophy of the supinator longus—the other muscles of the upper arm remaining normal—accompany the common form of "wrist drop" paralysis.

(3) *The Aran-Duchenne Type.*—This form is characterised by paralysis and atrophy of the small muscles of the hands, namely, the interosseous muscles and the thenar and hypothenar eminences. Atrophy is always well marked, and accompanies rather than succeeds loss of power. This type may be primary and the only manifestation of lead poisoning, but more frequently it complicates the classical type of paralysis. It is named from the close resemblance the condition and attitude of the hands bear to the type of progressive muscular atrophy described by Aran and Duchenne, and which is known to depend on degeneration of the ganglion cells of the anterior horns.

In some cases paralysis and atrophy are partial in distribution, and affect the abductor pollicis and the first dorsal interosseous muscle more than the other muscles. The preponderant affection of these muscles has been attributed to their over-use in particular occupations. Thus Moebius found it very frequent in file cutters, especially in those who with the left hand hold the chisel between the first phalanx of the thumb and the metacarpal bone of the index finger. In these persons Moebius observed that paralysis and atrophy started in the muscles of the left thenar eminence.

The accompanying engraving from a photograph represents the hand of a patient recently under my care, who in addition to extreme wasting and weakness of the extensor muscles of both forearms presented marked atrophy of the small muscles of the hands. The patient, a man aged

20, had worked for five years in a linoleum factory, and his work consisted in sprinkling a colouring mixture, one-sixth of which was composed of red lead, on to rollers. His illness began in October, 1891, with constipation and severe attacks of colic. He was able to attend to his work till February, when first his right and then his left arm gradually became weak. On admission to the Manchester Royal Infirmary on March 14th he presented the typical facies and gum line of lead poisoning.



With the exception of the supinator longus and the muscles on the front of the forearm, all the muscles of both arms below the elbow were weak and wasted. Thus, in addition to the ordinary type of paralysis, all the small muscles of both hands were greatly wasted and very feeble. There was no sensory disturbance. The knee-jerks were exaggerated, and the quadriceps femoris on both sides was slightly wasted.

(4) *Peroneal Type.*—Paralysis of the muscles of the lower limbs is not a common result of lead poisoning, and when present it either complicates paralysis of the arms or forms part of a more generalised paralysis. The muscles chiefly affected are the long extensor of the toes and the peronei, while the tibialis anticus, although supplied by branches from the same nerve viz., the external popliteal, usually escapes, just as the supinator longus does in the upper limb. Sensory phenomena often precede and accompany the paralysis. The patient may suffer from neuralgic pains in the knees and joints of the feet, or from numbness and tingling along the outer aspect of the legs. Sometimes there is hyperæsthesia either cutaneous or muscular, less commonly anæsthesia in the territory of the external popliteal nerve.

The patient affected with paralysis of this distribution is unable to stand on his toes, he walks on the outer side of his feet, and finds it difficult to get up and down a ladder or the stairs. In well-marked cases there is a distinct "high-stepping" gait.

This type of paralysis is, perhaps, more frequently met with in children who are the subjects of lead poisoning than in adults; at any rate, according to Putnam, who has studied lead paralysis in children, there appears to be a greater tendency in children than in adults for the muscles of the lower limbs to be selected by the poisonous effect of lead. In some cases the tibialis anticus is attacked in addition to the other muscles on the front of the leg. Remak relates a remarkable case in which there was paralysis of the deltoids, the extensors of the wrists and fingers as well as of the tibialis anticus and calf muscles, while the common extensor of the toes and the peronei were quite normal.

Laryngeal Paralysis.—In rare cases one or both vocal cords are paralysed and wasted. Sajous and Morrell Mackenzie mention paralysis of the adductors of the glottis, while Seifert shows that all the muscles of the larynx, either separately or in groups, may be paralysed by lead as well as by other toxic agents.

Generalised Forms.—In chronic cases of lead poisoning, when paralysis of the ordinary type has existed for some time, it occasionally happens that other muscles than those previously affected become involved, and paralysis slowly or quickly spreads to all the muscles of the limbs, and occasionally even to those of the trunk. Dejerine-Klumpke distinguishes three varieties. (1) Paralysis of slow generalisation. (2) Paralysis of rapid generalisation. (3) The febrile form of generalised paralysis.

In the first variety, paralysis is usually limited to the four extremities. In the second variety, all the muscles of a particular region are suddenly attacked, and may be reduced in a few days to a condition of absolute powerlessness. As in the first variety, the paralysis may supervene in old cases of localised lead paralysis, or it may be generalised from the outset, and pursue a descending or an ascending course. The patient lies on his back unable to move body or limbs; he cannot feed himself, and owing to involvement of the intercostals, diaphragm, and larynx, he suffers from intense dyspnœa and aphonia. The muscles of the head and neck are commonly spared. As a rule, most of the muscles quickly recover, but the extensors of the forearm and the small muscles of the hand may remain completely wasted, and paralysed for months or years. Recovery is the rule, and it is quite exceptional to see a patient die from slow asphyxia, as in severe cases of alcoholic paralysis. Yet death from respiratory paralysis has been observed, witness the following case recorded by Straus and Heugas:—

A man, aged 29, a painter on glass, after a severe attack of colic, suffered from painful cramps and a sensation of numbness in the hands. Twenty days later his legs became feeble, and he had numbness in the soles of his feet. There was no history of syphilis or of alcoholic excess. A month after the onset of paralysis his condition was as follows:—He was anæmic, but had no blue line on the gums. There was absolute paralysis of the four limbs, with marked atrophy of the muscles. The loss of power and wasting were as marked in the thenar eminence and in the flexors of the hand as in the extensors and deltoid. The patient was unable to dress or feed himself. He could not raise his arms above the level of the bed, and had great difficulty in performing the slightest movements with his fingers. Atrophy was also general; it was severe in the lower limbs, especially in the thigh muscles. As to the trunk, the only visible muscle was a fasciculus of the pectoralis major; the intercostal spaces were deep grooves. Intelligence and sensation were normal. There was no pyrexia; there was no girdle sensation; no bladder or rectal disturbance, and, in fact, no indication of spinal disease. A few weeks after the onset of paralysis the patient was carried into the Hôtel Dieu in a dying condition, and with symptoms of excessive difficulty in respiration. The diaphragm, however, acted well. Faradic contractility was abolished in certain muscles, as the extensors, and was notably diminished in others. The patient suffered from intense dyspnœa and oppression, symptoms which could not be explained by any pulmonary affection, and which were attributed by his doctors to a lesion of the bulbar region. His wife would not consent to his remaining in the hospital; he was removed in a dying state, and succumbed a few hours later to asphyxia.

Heugas remarks that the paralysis in this case was accompanied by rapid atrophy, and attacked the trunk muscles as well as the four extremities; there was abolition or diminution of electro-muscular contractility; a total absence of cerebral symptoms, of sensory disturbance, and of cutaneous trophic lesions; and the sphincters of the bladder and rectum were unaffected. Both he and Duchenne, who also saw the case, regarded it as an example of subacute general spinal paralysis.

Another case is recorded by M. Heugas in which repeated attacks of lead colic were followed by paralysis, first, of the extensor muscles of the forearm, and in a month of almost all the other muscles of the upper limbs, trunk, and lower limbs. The muscles underwent rapid atrophy, and their faradic contractility became abolished. The sphincters were intact. The only sensory symptoms were diffuse pains in the upper limbs.

Generalised paralysis with fever. One of the most constant characters of lead paralysis, whether of local or general distribution, is its evolution without pyrexia. There are, however, a few cases on record in which a universal paralysis was associated with fever. Meignen, who has written a thesis on this subject, says: The onset resembles that of adult spinal paralysis. The patient is suddenly seized with a rigor, accompanied by general malaise and violent pains in the head. There is nausea and vomiting. The tongue is dry and the skin hot. The temperature attains its maximum quickly, the period of ascent lasting only two or three days, to be followed by a fastigium of three to eight days, when gradual defervescence sets in, which, though less regular, somewhat resembles that of typhoid fever. The febrile attack which occurs at the onset of paralysis may be repeated, and then is usually the precursor of a further aggravation of the paralysis.

The symptoms of these generalised forms of lead paralysis closely resemble the subacute spinal paralysis described by Duchenne. But this designation was given to a group of symptoms which has no certain pathology. There is no post-mortem proof of its spinal nature, and the quick recovery testifies rather to a peripheral neuritis.

Cerebral Disturbance—Saturnine Encephalopathy.—Hysterical manifestations may develop from lead poisoning, just as they do under the influence of syphilis or other toxic agents. The hemiplegic weakness and hemianæsthesia that sometimes occur are probably of this nature. But the more frequent cerebral phenomena are delirium, coma, and epileptiform convulsions. These symptoms often occur at an early period of lead intoxication. Thus Oliver has observed them in young women who have worked only a few months amongst white lead. Sometimes they develop suddenly, and are the first indications of lead poisoning, but generally they are preceded by headache, giddiness, tremor, or dimness of vision. Optic neuritis often accompanies this acute cerebral disturbance, and death may occur from the severity of the convulsions or from the depth of the coma.

An attack of convulsions occasionally precedes the development of local paralysis, as in the following case reported by Putnam.

A woman, aged 27, began to fail in health and suffer from severe pains in various parts of the body, especially in the abdomen. After a time she began to have "faints," characterised by loss of consciousness, lasting two or three minutes, and preceded by slight dizziness and diffuse headache. These symptoms continued for about two years, and then the patient had a violent cerebral attack, with prolonged loss of consciousness, convulsions, and amaurosis. This was followed by another attack, after which the classical form of lead paralysis developed; this was succeeded by much local atrophy from which she never entirely recovered. The epileptiform attacks did not return after the appropriate treatment for lead poisoning was commenced.

Chronic general cerebral disturbance may succeed acute disturbance or develop gradually. Progressive mental failure also occurs, and it is stated that lead poisoning is one of the causes of general paralysis of the insane.

Ocular Symptoms and Affections of the Cranial Nerves.—Bilateral amblyopia may occur where there is no kidney affection, and without ophthalmoscopic changes. Sometimes it is transient, sometimes permanent. Two kinds of neuro-retinitis are met with, an acute and a chronic form. The latter occurs in old cases of lead poisoning in which the kidneys have become affected, and is identical with albuminuric retinitis. The acute form is characterised by great swelling of the disc and by the presence of hæmorrhages. The vessels are narrowed and often show white lines along their edges. These changes are often accompanied by loss of sight, which may be absolute. They may pass away under treatment, or set up optic atrophy. Optic atrophy is also met with apart from preceding signs of neuritis.

Paralysis of the ocular muscles has been occasionally observed in cases of lead poisoning. Buzzard has described a case of complete paralysis of the right third nerve in a patient affected with lead palsy, but the influence of syphilis could not be excluded. Oliver has observed diplopia, slight internal strabismus, and marked nystagmus in different cases. Putnam records the case of a girl aged 15½ who was poisoned with lead derived from drinking water. She suffered from dizziness, headache, and unsteadiness of gait. There was diplopia from paresis of both third nerves. Distinct ataxia in both upper and lower extremities was also present.

Inequality of the pupils and diminution or loss of the light reflex have also been observed.

Sometimes the facial nerve becomes affected.

I have once seen double facial paralysis in a painter, aged 28. He presented the characteristic signs of lead poisoning, and in addition to the facial paralysis there was extensive paralysis of both upper and lower extremities. The shoulder muscles, the triceps, biceps, peronei and flexors of the ankles were much atrophied, but the supinator longus and tibialis anticus were spared.

Morbid Anatomy.—The chief changes in lead paralysis are found in the muscles and peripheral nerves. The spinal cord is usually quite healthy, and the brain, even when cerebral symptoms have been severe, rarely presents decided changes in consistence or structure.

The muscles: Commencing atrophy is indicated by pallor; advanced atrophy by a yellowish appearance of the muscles. Under the microscope every change may be found between slightly wasted muscular fibres and empty sarcolemma sheaths; sometimes the sheaths contain fusiform flattened elements, imperfectly striated, which probably represent muscular fibres in process of re-formation. The wasting of the fibres is accompanied by an abundant multiplication of the nuclei of their sheaths, and these massed together in places give a moniliform appearance to the fibre. The connective tissue is slightly increased and often contains fat particles; the vessels, too, are thickened, especially the arteries, which show a certain degree of endo- and peri-arteritis.

The nerves: Morbid changes in the nerves are constant; they are most marked in the intra-muscular twigs, and become slighter the nearer to the cord the nerves are examined. The trunks, however, are not free, they present grey and rosy plaques, which are clearly separated from the white colour of normal parts, and are quite similar to the plaques found in the brain and cord in cases of multiple sclerosis. The changes in the peripheral nerves are considerable or slight in degree. The former constitute a parenchymatous neuritis, analogous to the "Wallerian" degeneration, namely, segmentation of the myelin, breaking up and disappearance of the axis cylinder, and proliferation of the nuclei and protoplasm of the sheath. The slighter changes, called "peri-axial neuritis" by Gombault, consist of segmentation of the myelin and multiplication of the sheath nuclei, but the axis cylinder is preserved; these alterations are sometimes segmental—that is, are limited to certain short tracts of the nerve, the intervening portions being healthy. These degenerative changes, while constantly found in the peripheral branches, and frequently in the nerve trunks, are rarely present in the anterior roots.

The spinal cord: In the great majority of cases of lead paralysis, an examination of the cord has given negative results. In a few cases atrophy of the ganglion cells of the anterior cornua has been found, but in some of these cases the area of atrophy did not correspond to that of the affected muscles, while the condition of the latter could be fully accounted for by the presence of degenerative neuritis.

Perhaps the most satisfactory case in favour of the occasional spinal origin of lead paralysis is one published by Remak, the autopsy being made by Oppenheim. In this case the patient had suffered from the classical form of paralysis for some time, also from paralysis of the muscles innervated by the anterior crural nerve, as well as of the gastrocnemius and tibialis anticus. He succumbed during an attack of lead encephalopathy and gangrenous stomatitis. The post mortem revealed interstitial nephritis, cardiac hypertrophy, a recent hæmorrhagic focus in the right temporal lobe, and a cystic focus outside the head of the right corpus striatum, together with the usual degenerative changes in the muscles, and in the musculo-spiral and external popliteal nerves. The cord lesions occupied the grey substance, the cervical and lumbar enlargements being more affected than other parts. The lesions consisted in disappearance of the ganglion cells, diminution of the nervous fibres in the grey substance, with overgrowth of the neuroglia; the vessels were increased in number, and many were dilated and had thickened walls. The anterior roots were normal, both in the cervical and lumbar regions.

Pathology.—Leaving out the question how far all forms of peripheral neuritis depend on central influences, there is abundant proof that lead paralysis and degenerative neuritis are found in constant association, and often when the cord and brain appear to be quite healthy.

Lead is remarkable in picking out motor nerves, and almost exclusively those which supply extensor muscles. This peculiar selective influence is most strikingly shown in the common wrist-drop form of paralysis, where the posterior interosseous branch of the musculo-spiral nerve is alone attacked. Extensor muscles are most affected in other forms of peripheral neuritis, but not in the same exclusive manner, nor are sensory fibres so frequently spared as in the case of lead neuritis. Without attempting to offer any hypothesis for the explanation of peculiarities in the selective action of different poisons, it is desirable with respect to lead paralysis to bear in mind: (1) That the presence of sensory disturbance is not so rare as text-books would lead one to suppose; for (*a*) in the peroneal type of paralysis, diminution of cutaneous sensibility is probably the rule; (*b*) the presence of normal cutaneous sensibility is probably as common in traumatic lesions of the musculo-spiral nerve as in the classical type of lead paralysis; also, in the latter, transient patches of anæsthesia are not uncommon if carefully looked for, and diminution of the faradic sensibility is very frequent. (2) That the strict localisation of the influence of lead to the extensors of the forearm is by no means invariable even in the ordinary type. Several times I have found the supinator longus on one or both sides to be affected, and often a careful examination of the lower limbs has shown some change either in the condition of the knee-jerks or in the nutrition and strength of some of the thigh or leg muscles.

But as Dr. Gowers points out, the action of lead is certainly not confined to the peripheral nerves. It is probable that the cerebral symptoms are due to the direct action of lead on the nerve elements of the brain, and as to the cord we have already mentioned, that it has shown definite lesions in several cases of lead paralysis. Dr. Gowers believes that degeneration of the ganglion cells of the anterior horns is related chiefly to cases in which muscular atrophy and paralysis slowly progress side by side, the former accompanying rather than succeeding the latter. This atrophic form may be studied in the Aran Duchenne type of lead paralysis, where the intrinsic muscles of the hand are mainly involved. In connection with Dr. Gowers's view, a case reported in great detail by Madame Dejerine-Klumpke (*loc. cit.* p. 108) requires a brief notice. In this case, in addition to paralysis and atrophy of the extensors of the forearm, there was marked atrophy of all the small muscles of the hand; yet at the post-mortem examination the spinal cord was found to be quite healthy, while extensive changes were found in the nerves of the brachial plexus and their terminal branches.

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Chabbert (L.) "Paralysie saturnine et nevrites périphériques."—*Echo méd.* (Toulouse) 1891, 2 S., V., 553.

Anders (J. M.) "Nervous forms of lead poisoning, etc."—*International Clinics* July, 1891.

(*b*) Arsenical Paralysis.

Paralysis is an occasional result of poisoning by arsenic. The common type, which usually occurs in cases of acute poisoning, closely resembles the paralysis due to chronic alcoholism. As a rule the lower extremities are attacked before the upper; in both cases the extensor muscles of the hands and feet are principally implicated, so that the patient cannot properly extend his fingers, toes, wrists, or ankles, and in some cases there is complete wrist and ankle drop. Atrophy of the muscles occurs very quickly, probably more quickly than in the peripheral palsies of lead or alcohol. The muscles respond badly to the faradic current, and often show the qualitative alterations to galvanism which characterise the reaction of degeneration. The paralysis may spread to the small muscles of the hands and feet, or to the arms and thighs, and occasionally may become almost universal in distribution.

Tremors, general or localised, may be present, and spasmodic movements have also been described. Thus Pick observed spasmodic flexion of the great toes, while in a remarkable case of arsenical paralysis recorded by Kovàcs the fingers of both hands were the seat of movements like those of athetosis. These consisted in slight flexion and extension, and less frequently ab- and adduction of all the fingers. The movements only occurred when the limbs were in a position of rest and ceased directly during the performance of voluntary movements. In Kovàcs's case there was no tremor.

Arsenical paralysis is ushered in and accompanied by marked sensory disturbance. The patient complains of severe darting, smarting, burning, or rheumatic-like pains in the limbs, and of numbness and tingling in the fingers and toes. The spontaneous pains are often severe enough to prevent sleep. Painful cramps are also present, and there may be much cutaneous and muscular hyperæsthesia. These irritative phenomena are quickly followed or accompanied by diminution or loss of the cutaneous sensibility. The muscular sense is also frequently affected, so much so sometimes as to constitute a true ataxia. As a rule any inco-ordination of movement can be explained, or is overshadowed, by muscular weakness; but occasionally it is the dominant feature of the case, which then closely resembles one of locomotor ataxia. Hence Dana distinguishes two types of arsenical paralysis :—

"(1) The ordinary mixed motor and sensory paralysis, the motor troubles and atrophy being more marked. (2) The pseudo-tabetic form in which there is no pronounced motor paralysis, but marked sensory troubles, and especially ataxia."

The latter type is stated by Kovàcs to be more common in cases of chronic than acute intoxication. The tendon reflexes are almost invariably lost, while the superficial ones are usually present. In the only case of arsenical paralysis that has come under my own observation, the knee-jerks and plantar reflexes were both increased; the other symptoms of the case were pricking sensations in the hands and feet, weakness of the muscles on the front of the legs, relative anæsthesia of the hands, feet, and outer side of the legs, and hyperæsthesia of the calves. The patient, a man aged forty-one, had worked in arsenic three months; he was only seen once, so that the subsequent history of the case cannot be given. It seems probable that the deep reflexes are often exaggerated in the early stages of arsenical as in alcoholic and other toxic palsies, but Kovàcs holds the contrary opinion, namely, that their loss is much more constant when the paralysis is due to arsenic than to other poisons.

The course of arsenical paralysis is similar to that of alcoholic paralysis, and its duration varies, according to the intensity of the intoxication, from several weeks to several months.

Its clinical history justifies the assumption that the peripheral nerves are implicated rather than the spinal cord. But the anatomical proof of this is not yet fully established. Jarschka, in 1882, was the first to suggest that the paralytic symptoms depended upon a multiple neuritis, and, according to Putnam, this view has been confirmed by the autopsy of a case reported in the *Canada Medical and Surgical Journal*, 1886, p. 716. Quite recently Erlicki and Rybalken examined the nervous system in two cases of arsenical paralysis, and found disease of both the anterior horns and peripheral nerves. They believe that the degeneration of the spinal ganglion cells, which is sometimes found in toxic cases, is due to physiological and chemical peculiarities of the blood, and not to the direct effect of the poison on nerve elements. It has also been shown that arsenic injected subcutaneously is capable of destroying adjacent nerves, apart from any changes in the nerve centres. The careful experiments of Vrigens in 1881 make it probable that no part of the nervous system is exempt from the influence of the poison. Hæmorrhages widely distributed in the brain and cord are sometimes found, and are thought to result partly from paralysis of the vaso motor nerves, and partly from changes in the constitution of the blood and vessels.

Popoff has produced experimentally myelitis more or less diffused; and his examination of the cord of a man who was acutely poisoned by arsenic confirms his experimental observations on dogs.

The distinction of arsenical from alcoholic paralysis is based on the absence of a history of intemperance and on the discovery of a cause of arsenical poisoning, as well as the presence of associated symptoms which are known to be the result of poisoning by arsenic. These are (1) certain skin lesions; (*a*) a peculiar pigmentation, sometimes closely resembling that of Addison's disease; (*b*) herpes zoster, which is present in a small percentage of cases; (*c*) bullous or erythematous eruptions; (2) loss of hair; falling off of the nails; (3) intermittent dysuria or glycosuria; (4) œdema of the eyelids; (5) ulceration of the gums and fauces; (6) the presence or history of acute unaccountable attacks of indigestion associated with nausea, salivation, and epigastric pain.

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HEEKENLAUER (G.). "Ueber Arsenikal-Muskelatropie."—Würzburg, 1883.

KOVÁCS (F.). "Ein Fall von Arseniklähmung."—*Wiener klin. Wochenschr.*, 1889.

M'CLURE (H.). A case of multiple neuritis due to chronic arsenical poisoning."—*Lancet*, 1889.

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PUTNAM (J. J.) "On chronic arsenic poisoning, etc."—*Boston Med. and Surg. Jour.*, 1889.

PUTZEL (L.). "A case of arsenical paralysis; multiple neuritis."—*New York Med. Rec.*, 1888.

SCOLOBOUZOFF. "Paralysie arsenicale."—*Arch. d. Phys. Norm. et Path.*, 1887.

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WOOD (H. C.). "A case of arsenical neuritis."—*New York Rec.*, 1888.

BARTON (S.). "Two cases of arsenical peripheral neuritis."—*Lancet*, 1890, II.

MARIK (S.). "Des paralysies arsenicales."—*Wien klin. Wochenschr.*, 1891, 31—40. (These papers contain a good bibliography.)

CUTLER (E. G.). "A case of multiple neuritis, due to arsenic."—*Boston Journ.*, 1890

PAL. "Ueber multiple Neuritis."—Wien., 1891. Beobachtung VIII.

FEILCHENFELD. "Neuritis multiplex cum Glycosuria."—*Deutsche med. Woch.*, 1890.

ERLICKI und RYBALKIN. "Ueber Arseniklähmung."—*Arch. f. Psych. und Nervenkrankh.*, Bd. XXIII., S. 861.

(*c*) MERCURIAL PARALYSIS.

The most common and characteristic nervous symptom of poisoning by mercury is tremor. This at first occurs only on movement, and in this respect resembles that of disseminated sclerosis, but the latter is coarser and more irregular. Ultimately the tremor may become constant, and then resembles that of paralysis agitans. Frequently limited to the head, face, tongue, and arms, it may spread to the lower limbs and to other parts of the muscular system, while in severe cases it may interfere with articulation, mastication, or even respiration. At first muscular power is maintained, but after a time the limbs become weak, and sometimes there is distinct paralysis.

Kussmaul describes a case in which permanent paralysis of the right arm became developed after severe mercurial tremor and clonic spasms. Letulle, who has written an important paper on the subject of mercurial paralysis, points out that the weakened muscles are lax, but that distinct atrophy is rare. The tendon reflexes, normal in some cases, are diminished in others. The paralysed muscles give normal reactions to the faradic and galvanic currents. The sphincters are unimpaired. Sensory disturbances accompany the paralysis; there may be various paræsthesiæ, scattered patches of anæsthesia, together with hyperæsthesia of the skin and also of the special senses.

Letulle's experiments on dogs and guinea-pigs tend to show that the subcutaneous injection of mercury causes degeneration of the nerves in the immediate neighbourhood. The nerve fibres presented a progressive segmental destruction of the medullary sheath, without nuclear proliferation and with unaffected axis cylinders. Letulle lays stress on the limitation of the effects of mercury to the nerve medulla, and on the rapid regeneration which takes place and which, in his opinion, corresponds to the speedy recovery from mercurial paralysis in man.

KUSSMAUL. "Untersuchungen über den constitutionellen Mercurialismus."—Würzburg, 1861.

Letulle (A.). "Recherches cliniques et expérimentales sur les paralyses mercurielles." *Arch. de Physiol norm. et pathol*, 1887, Nos. 3 et 4.

Letulle (M.) "Recherches expérimentales sur l'intoxication mercurielle."—*Comptes rendus*, 1887.

(*d*) PHOSPHORUS AND SILVER.

Paralyses have been observed after both acute and chronic poisoning by phosphorus, but their anatomical basis has not been fully investigated.

As regards silver, Dr. Gowers says that he has been unable to find any recorded instance of paralysis in man, but he relates the case of a gentleman, who had been ordered a dinner pill containing silver, which he took, on and off, for a period of twelve years. When seen by Dr. Gowers "the tint of the skin was very characteristic; his gums presented a well-marked black line, which resembled that of lead in being present where there was tartar, and absent, where the gum was in perfect apposition to the teeth. In both arms there was paralysis of the long extensor of the fingers and of the extensors of the phalanges of the thumb, not of the extensor of the metacarpal bone of the thumb. On the right side there was also paralysis of the radial extensors of the wrist. The affected muscles were wasted, and their irritability to faradism was lost; that to voltaism was preserved and increased. There was no affection of the legs."

II.—TOXIC MULTIPLE NEURITIS. (4) THE NEURITIS CAUSED BY ENDOGENOUS POISONS.

(*a*) PERIPHERAL NEURITIS IN ACUTE RHEUMATISM; IN CHOREA; IN RHEUMATOID ARTHRITIS; ALSO THE RELATION OF MUSCULAR ATROPHY TO AFFECTIONS OF THE JOINTS.

The fact that any inflammation of a joint is almost invariably attended with rapid wasting of the muscles that move the joint, was first definitely described by John Hunter, in 1836. He recognised, too, that the atrophy is not the result of default of movement, for he says, when the other limb is examined, it is seen that the muscles there have preserved their volume almost entirely, although they have not been more moved than the muscles of the diseased limb. Hunter also noted that the muscles were sometimes paralysed, even when they were not atrophied.

Gallavardin. "Les paralysies phosphoriques."—Paris, 1865; et *Gaz. méd. de Paris*, 1864, p. 6, *et seq.*

Kreyssig (F.) "Ueber die Beschaffenheit des Rückenmarks bei Kaninchen und Hunden nach Phosphor und Arsenik-Vergiftung, nebst Untersuchungen über die normale Structur desselben."—*Virchow's Arch.*, Bd. CII.

Gowers. Vol. I., p. 887.

In 1869, Ollivier, in his thesis on muscular atrophies, alludes to a case under the care of Duchenne, in which a traumatic synovitis of the knee was followed by great wasting of the thigh muscles, and he suggests the reflex nature of the phenomenon, and adds that such muscular atrophy is also met with in chronic articular rheumatism.

Vulpian also, in 1875, pointed out that the wasting begins almost as soon as the arthritis, and he ranges it amongst reflex atrophies. Fort, in 1876, showed that a slight arthritis of the shoulder may be accompanied by such rapid atrophy of the deltoid, that in eight days the flattening of the shoulder may simulate the deformity due to dislocation.

Sir James Paget, in his clinical lectures, also speaks of the extreme rapidity with which this muscular atrophy supervenes in all acute articular inflammations. But the most complete account of the subject is to be found in a thesis by Valtat, published in 1877. He points out the importance of noticing that although it is difficult to separate paralysis from atrophy, yet sometimes the former is present without the latter; thus he mentions a severe sprain of the wrist where, after the pain and swelling had gone, there was found complete paralysis of the extensor muscles, while the atrophy was absent or doubtful.

The muscular atrophy, he says, is always marked and distinct at the beginning of the second week; thus, between eight and eleven days after a knee arthritis, the thigh on the affected side will measure about one inch less than the healthy thigh.

Pain is not essential as an antecedent, for an indolent hydrarthrosis may be followed by atrophy; and, conversely, Paget has observed wasting consecutive to a neuralgic joint pain without inflammation.

The extensor muscles are the most profoundly and the first to be affected. Thus, the quadriceps, when the knee is inflamed; if the hip, the glutei chiefly waste; if the shoulder, the deltoid; if the elbow, the triceps. But it is not uncommon, Valtat says, especially in old cases, to see other muscles affected, even an entire limb, although only one joint is inflamed.

Valtat obtained similar results by experimenting on dogs. He injected irritating substances, as ammonia, into the joints, and the resulting arthritis was always very intense, and the ensuing atrophy rapid and extensive, involving the whole limb in a few days; but here, also, the extensor muscles showed the greatest wasting.

Valtat, in seeking for an explanation, rejects disuse on account of the rapidity and degree of the wasting, and shows, too, that the effect cannot be due to any local inflammation of the muscles or nerves passing to them from the affected joints, for the whole length of a muscle suffers equally; moreover, he found no signs of inflammation on microscopical examination of the wasted muscles, resulting from the traumatic arthritis produced by experiment, and he finally accepts Vulpian's reflex theory as the correct one, viz., that the irritation of the articular nerves so alters the nutrition of the ganglion cells of the cord as to seriously enfeeble the activity of the motor fibres derived from these cells.

Lastly, Charcot re-opens the question in clinical lectures delivered in 1883, and, while we acknowledge Valtat's thesis as the most complete account of the subject, and accord to Hunter the honour of the discovery, it is to Charcot, I think, that we feel particularly indebted for the dramatic force, and marvellous lucidity, with which he has set the picture before us. He relates the case of a man, aged 23, who, about a year prior to examination, struck his right knee in jumping over a fallen tree. The injury did not appear to be severe, for he walked several miles without difficulty; at length he stopped for a time, and then found himself unable to walk without a stick. For a week afterwards he kept his bed; the joint was swollen, though not very painful, but there was a remarkable loss of motor power in the limb. Two things were evident and striking, there was paresis and atrophy of all the muscles of the limb, but both paresis and atrophy were most marked in the quadriceps extensor. Tested by electricity, the muscles and nerves showed great quantitative diminution to both galvanic and faradic currents, but no qualitative change was obtained, showing that the condition was one of simple, and not degenerative, atrophy. Charcot obtained lively muscular contractions by the application of the electric spark from the frictional machine. The knee-jerk was increased not only on the affected side, but on the other also. Charcot, after considering many hypotheses, finds himself obliged to admit the existence of a spinal affection, but in the absence of the reaction of degeneration, he cannot admit any serious change in the anterior horns of grey matter, and is reduced to the suggestion of a kind of stupor or inertia of the nerve cells. He explains the exalted knee-jerks, by supposing that while the cells of the affected region are in a state of torpor, there is a condition of increased reflex excitability in the rest of the cord. He relates also a case of chronic rheumatoid arthritis, affecting many joints, in which, although there was neither pronounced inflammation nor pain there was rapid wasting, and loss of muscular power. There was the same loss of electric sensibility as in the other case, except that the right vastus externus gave the reaction of degeneration.

A patient in the Manchester Royal Infirmary under the care of Dr. Simpson showed in the most typical manner the wasting of thigh muscles which so many authors have noted in affections of the knee. The patient was a man aged 20 years, the subject of subacute rheumatism. On admission, in addition to pain and slight swelling of some of the joints, there was considerable effusion into the right knee;

the thigh muscles felt distinctly smaller and softer than those of the left thigh, and when measured, two days later, or nine days from the time the knee first became painful, the right thigh was nearly one inch less in girth than the left one. The wasting appeared to affect all the muscles, and the adductors felt as soft as the quadriceps.

Charcot points out that in other cases of articular disease, spasmodic contraction, and not atrophy, is the dominant feature, as *e.g.*, in acute hip disease, and he refers to his description, given thirty years ago, of the deformities met with in rheumatoid arthritis, and he still looks on them as mainly produced by spasmodic contraction, developed by reflex action from the joint irritation. But when we see the deformities, he says the spasmodic contraction has usually ceased long before; the deformity, however persists, owing to the thickening of the tissues, the partial dislocations, and the shortening of the ligaments.

Now, with regard to this early spasmodic contracture in rheumatic cases, the evidence is not forthcoming in the large majority of patients. Thus, one may follow out a case of acute rheumatism, and see a chronic deformity of the hand, for example, result without ever being able to detect a stage of contracture of muscle. Now, as in all such cases atrophy of certain muscles is met with, the deformity may, perhaps, be more correctly attributed to the *normal*, not spasmodic, unopposed action of healthy muscles, their antagonists being weakened and wasted; and, indeed, unless we see these deformities at a very late stage, it is usually pretty easy, so far as my experience goes, to reduce them; the joints of the fingers are generally freely movable, and can be put into normal positions.

At the same time it is true that in some cases spasmodic contraction of muscles may be observed at the commencement of a rheumatic attack. Thus, quite recently a woman came to the out-patient department complaining of severe pain in the left hand. On examination the wrist and knuckles were found to be tender and slightly swollen, and the fingers were flexed; the flexion was considerable at the two proximal and slight at the distal joints; it was also rigid flexion, the posture being not merely an instinctive one for the relief of pain, but one due to spasm of the flexor muscles.

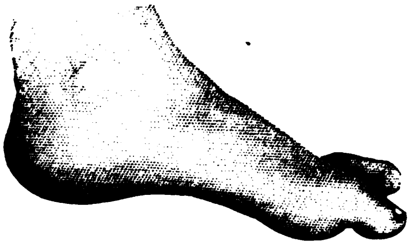
It is doubtful whether the reflex theory affords an adequate explanation for the amount of atrophy often met with in the neighbourhood of joint affections. Surely, the stupor or inertia of the ganglionic cells cannot persist for months in a degree sufficient to produce such atrophy—say, of the thigh, that the femur may be readily grasped—without any organic change in the nervous system. It has been suggested by Ross, Erb, and others, that a depressed state or nutritional change of the central trophic cells may start degeneration, not of the anterior roots, but in the terminal fibres of the peripheric nerves. I may mention too that Moussons, who repeated the experiments of Valtat and then subjected the muscles and terminal nerves to a careful microscopical examination, found, in a few cases, distinct degeneration of nerve fibres; but it must be confessed that the change was inconstant, and appeared to bear no proportion to the degree of muscular atrophy.

The subject demands re-investigation, for while we may admit the reflexly-produced torpor of the ganglionic cells as a reasonable explanation of the sudden onset of the weakness and atrophy, it does not seem adequate to account for the progressive muscular atrophy which goes on long after the joint has completely recovered; it is difficult to believe that such persistent symptoms can depend on any mere functional derangement. We know little of the pathology of the terminal axis cylinders, with their motor end plates; possibly, in the future, it may be discovered that degeneration of these nerve elements may be responsible for many general atrophies now thought to be primarily and entirely disease of muscle tissue, or cases of so-called "simple muscular atrophy."

Having now cleared the ground by a consideration of what is common to all joint affections, we may turn to the muscular atrophies that cluster round a rheumatic attack, and which by their distribution and associations appear to have a special pathology.

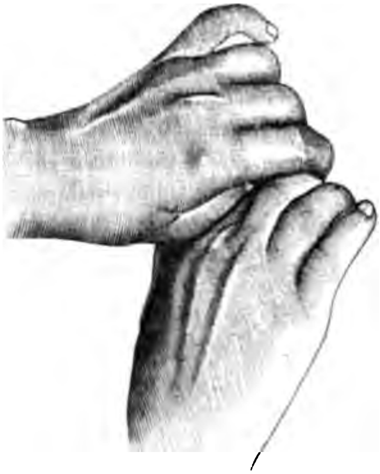
The following cases illustrate some of the chief varieties of paralytic phenomena met with in association with rheumatism. Some of them were under the care of my colleagues, Drs. Morgan, Simpson, and Dreschfeld, to whom I am indebted for permission to give the following abstracts:—

Case 1.—Kate A., 23 years, admitted under Dr. Dreschfeld March 1, 1888. Her illness began early in January with pain and stiffness in the joints of the lower limbs. In February, last year, she had a similar, though less severe, attack of rheumatism. Prior to this she had fairly good health. A sister died æt. 27 years, of rheumatic fever and heart disease; her other sisters and brothers and her parents are quite healthy.



On admission she was pale, sweating profusely, and had much pain in the limbs. In the lower limbs there was effusion into both knee joints; the ankles were very tender and slightly swollen. There was also tenderness on pressing over the external popliteal and posterior tibial nerves, together with some general hyperæsthesia of the skin and muscles. The feet were unduly extended at the ankle and there was a slight droop of the great toes, the other toes of the right foot were natural in appearance and position, but those of the left foot were hyper-extended at the metatarso-phalangeal joints and flexed at the other phalangeal joints, giving a somewhat claw-like appearance to the foot, and the dorsum of this foot, near the toes, was sunk, as compared with the right foot, and the muscles of the left leg were softer and more wasted than those of the right leg. On testing the cutaneous sensibility with pin prick and finger touch, distinct, though not absolute, anæsthesia was present on the inner side of the right great toe and foot and over the inner half of the sole, and it extended up on the inner side of the leg about halfway to the knee, the area corresponding roughly to the distribution of the internal saphenous nerve.

As to the upper limbs, the right elbow was semi-flexed, stiff, painful on movement, and sore to palpation, especially in the ulnar groove, where around the nerve a gelatinous sort of swelling was felt. The hand



was midway between supination and pronation, and movement in either of these directions was very limited. There was a slight swelling on the back of the right wrist. The knuckles were sore and slightly swollen. The fingers were flexed at the metacarpo-phalangeal joints, and the patient could not extend them; there were depressions between the metacarpal bones on the back of the hand, owing to wasting of the interossei, the grooving being especially marked in the fourth space and in the first, where the muscular mass between the metacarpal bones of the thumb and index finger felt soft and thin. The hypothenar eminence was also wasted. She could not oppose the thumb against the tip of the little finger, and ad- and ab-duction of the fingers were feebly performed. There was a slight diminution of sensibility over parts of the right hand. The left arm and hand were affected in a similar manner. Well-marked tremor of the hands and fingers was observed when they were held out, and occasionally twitching of the forearm muscles was seen. The tendon reactions in the arms and legs were increased. The heart and other organs were normal.

Two days later the partial anæsthesia in right leg and hand had disappeared.

On March 15th a slight systolic apex murmur was heard for the first time.

On April 2nd a crop of subcutaneous nodules was found over the external condyle of the left humerus, varying in size from a pin's head to a hempseed; there were also single nodules on the knuckles of the index and middle fingers of both hands. These nodules were very painful on pressure. There was still much joint pain and sweating, and the tendon reactions were again found to be increased. The atrophy of the interossei, greater than formerly, was especially noticeable in the 1st, 3rd, and 4th spaces. The little fingers were more flexed at the two terminal joints, and still hyperextended at the first. The forearm extensors appeared to be wasted. Both ulnar nerves were tender to pressure. On testing the interossei with electricity, they showed diminished reactions to both the faradic and galvanic currents, but probably no qualitative change to galvanism.

During the month of May the patient remained in much the same condition. The nodules at left elbow, however, became much larger. It was also noticed that the lower ends of both ulnar bones were distinctly thickened near the styloid process. Ultimately she made a fair recovery.

In this patient then, suffering from an attack of acute rheumatism, as proved by the articular affection, the sweating, the pyrexia—varying from 99° to 102°—the crops of subcutaneous nodules and the existence in all probability of endocarditis, we have (1) tenderness and swelling over the ulnar nerves with atrophy and paresis of the abductor indicis and other interrosseous muscles supplied by these nerves; (2) probably similar atrophy of the interossei of the left foot supplied by branches from the posterior tibial; (3) temporary anæsthesia in the region of the internal saphenous nerve.

Case 2.—Another patient under Dr. Dreschfeld's care, who had a severe attack of acute rheumatism accompanied by endocarditis, pericarditis, and pleuro-pneumonia, also presented marked wasting of the interossei. The hands were good examples of what might be called the typical rheumatic hand. Thus, when at rest, the metacarpal portion looked concave, the knuckles forming a prominent anterior barrier of this shallow hollow, and the wrist the gradually sloped posterior boundary. The general concavity was interrupted (in this patient) by the extensor tendons of the middle and ring fingers. The concave metacarpus was well seen by looking at it from the ulnar side; then, instead of a straight or slightly convex line as in health, a concave one was seen, which was especially marked over the first space, owing to atrophy of the abductor indicis.

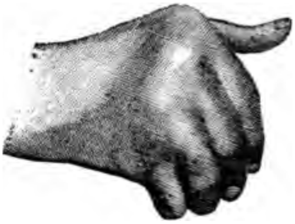
In this patient there was also slight thickening of the left ulnar nerve. There was distinct wasting of the muscles on the front of the legs, and purpuric spots were present on the outer sides.

Case 3.—A lady, under my own care, with rheumatic fever, which began in December, 1886, and was very severe and complicated by pericarditis and left pleurisy. Almost at the outset she had partial anæsthesia in the distribution of the ulnar nerve on one side, and also along the inner side of the same arm, corresponding to the distribution of the internal cutaneous nerve: this anæsthesia lasted three or four days. There was wasting of all the thumb muscles and of the interossei; and the terminal phalanges of the fingers were slightly *hyperextended;* this made me think that the deep flexor of the fingers, which is supplied by both median and ulnar nerves was affected, and certainly there was evidence in the thumb and interossei muscles that both nerves were implicated.

Case 4.—Another patient of mine, a man aged 70, had severe erysipelas which started in the right leg, and subsequently attacked the face. During convalescence he had what looked like an attack of rheumatism; the left elbow became very swollen and painful, and thickening round the ulnar nerve could be felt. All the muscles of the forearm and hand began to waste very rapidly, and showed well-marked fibrillary tremors, the fibrillation of the extensor muscles being extreme, and in a few days atrophy of these muscles was advanced. A little later the right elbow and forearm were affected in precisely the same way. It looked as if the rheumatic swelling about the elbow joint had pressed directly on the median, musculo-spiral, and ulnar nerves; or as if there were a separate inflammation of the sheaths of these nerves.

Case 5.—The following are notes of a man suffering from subacute rheumatism, who was under the care of Dr. Simpson:—"Both forearms exhibit fibrillary tremors of the flexor and extensor muscles. The grasp is weak, especially of the left hand; all the muscles of the *right* upper extremity are weak, the strength of the left being in marked contrast to them; this is especially noticeable in the triceps and supinator longus. Thickening is to be felt around the right ulnar nerve, and the interossei are decidedly atrophied. All movements of the fingers are weak and performed with much tremor, and opposition of the thumb is impossible, nor can the hand be completely closed. In the left leg there is evident diminution to cutaneous sensibility in the area supplied by the internal saphenous nerve. The external popliteal, while distinctly thickened on the right side, feels normal on the left. The feet show hyperextension of the toes at the first and flexion at the terminal phalangeal joints, and he cannot, by a strong voluntary effort, flex the proximal joints. All the tendon reactions are slightly increased." The patient remained in the Infirmary one month, then went to the Convalescent Hospital at Cheadle. I saw him on his return in a few weeks, and though the hands were plumper, there was still distinct grooving between the metacarpal bones. Here then we have paralysis, atrophy, and fibrillation of muscles supplied by the musculo-spiral, median, and ulnar nerves, together with anæsthesia of the skin supplied by one internal saphenous nerve.

Case 6.—Another female patient under Dr. Dreschfeld had the chronic deformity of the hand seen in the woodcut, and this dated from an attack of rheumatic fever two years ago. When in the hospital she had pain and slight swelling in several joints, and her temperature was occasionally raised. In this case there were several curious disturbances of cutaneous sensation; thus an area of well marked though not absolute anæsthesia over the ball of the right great toe, another extending along the outer side of the left leg from the malleolus half way up the fibula, and there was much hyperæsthesia of the soles of the feet and paræsthesia over the plantar surface of the toes of the left foot. In the right upper extremity there was complete anæsthesia over the palmar and dorsal surface of the hand and fingers, partial anæsthesia over the thumb and the inner two-thirds of the anterior aspect, and over the back of the right forearm. The cutaneous sensibility was also slightly



diminished over the upper arm. The anæsthesia varied in intensity, and was of temporary duration. The muscles of the right upper were all much weaker than those of the left upper extremity.

In this case there was a suspicion that the anæsthesia was a hysterical phenomenon, but it was not so profound or so abruptly limited as is usual in that variety.

Another rheumatic case presented slight anæsthesia of the thumb and first finger. In another, a subacute case, there was partial ulnar anæsthesia and diminished sensibility also of the skin on the outer side of the left leg.

In most of the preceding cases the spine was examined and found to be free from tenderness; but there is another group of cases which I believe to be far from uncommon, in which, with certain parts of the spine tender to pressure, there is evidence of pressure on or irritation of the roots of some of the spinal nerves. The difficulty in such cases is to be sure that the presumed external pachymeningitis is secondary to a true rheumatism, and not primary, with associated joint pains from the neuritis. I will mention two:—

The first (*Case 7*), a man, æt. 40, under my care eight years ago, gave a history of chronic rheumatic joint pains. When I saw him he had no articular affection beyond some stiffness in the joints of the lower limbs, but there was a crop of subcutaneous nodules around the left patella. He had severe pain across the lower part of his back, and shooting pains down the sciatics, together with much tenderness over the sciatic and other nerves in the legs. There was also much aching along the right arm, and I found the cutaneous sensibility markedly impaired along the inner side of the little, ring, and middle fingers, and the inner side of the palm, forearm, and arm; there was also some numbness over the suprascapular fossa, and hyperæsthesia of the seventh cervical and first dorsal spines. Two days later the anæsthesia had gone.

Judging from his rheumatic history, and from the presence of rheumatic nodules, I thought it not unreasonable to suppose that the rheumatic poison had set up a slight inflammation of the dura mater, and possibly of the sheaths of the eighth cervical and first dorsal nerves.

Case 8.—The following notes refer to a patient aged 42, who was admitted to the Royal Infirmary, April, 1888, under the care of Dr. Dreschfeld. "He had rheumatic fever two years ago and has never been free from pain since. He was an in-patient for the first time last August, and then his ankles were swollen, and he had much pain down the spine. The muscles of the right upper arm were very weak, and the grasp of the right hand was extremely feeble. In September it was noted that he had severe pains in the knees, ankles, and great toes, but from the first the most severe pain has been down the right arm. At the present time all the movements of the right upper limb are weak, although the limb is as large and muscular-looking as the left one. There is slight numbness along the radial border of the right forearm. The spine is tender about the sixth and seventh cervical vertebræ, and to a less degree lower down, and the cutaneous tissues are sore to pinching from the spine radiating towards the shoulders. There is distinct thickening about the right ulnar nerve, and probably also of the right median nerve. Both hands and feet exhibit the condition known as erythromelalgia," and, I believe that a slight degree of erythromelalgia of the feet is not a very rare phenomenon in articular rheumatism.

The tender spines, with hyperæsthetic radiating tracks to the shoulders, together with weakness of the right upper, point to pressure on nerve roots; and the thickening of the median and ulnar nerves, with the condition of the hands and feet, to a neuritis; and I think we have fair grounds for looking on the case as a rheumatic one.

A third, but rarer, group of cases is of great interest, suggesting, as it does, that a general progressive muscular atrophy occasionally starts from a genuine attack of articular rheumatism. This is an old notion; for example, Sir William Roberts in his book on "Wasting Palsy," mentions a thesis by Thouvenet, who strangely enough contends that progressive muscular atrophy is primarily located in the peripheral nerves, and that it must be classed with rheumatic affections.

Case 9.—Several years ago I saw an example of this in Pendleton—a woman who had a severe attack of rheumatic fever, which laid her up fifteen weeks and was followed by general muscular wasting. I remember seeing her much wasted, with considerable articular deformity, and the right hand presented a marked claw-like position.

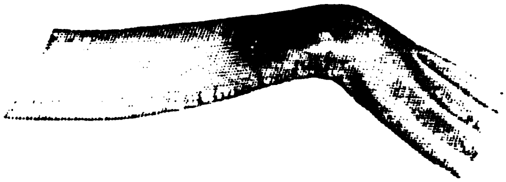
Case 10.—A man, aged 44, under the care of Dr. Morgan, who had had several attacks of rheumatism during the last 20 years. In October, 1887, he was feverish, sweated much, and many joints were painful and swollen, and the doctor in attendance said he had rheumatic fever. He began to get thin afterwards, and has wasted ever since. In January, 1888, there was general and great muscular atrophy and fibrillation of many muscles. There was also some irregularly distributed anæsthesia on the right forearm, and the front of the right leg. All the tendon reactions were slightly increased. A mitral systolic murmur was heard, and was conducted round to the angle of the scapula. There was very evident enlargement of the ends of some of the long bones; thus, at the elbow, the bones felt much thickened, and this was especially noticeable in connection with the head of the radius. Down each side of the front of the chest, and corresponding in appearance and position to the rickety beads met with in a young child, there was a remarkable row of bony nodules, apparently due to thickening of the ribs. These little knobs were certainly not the relics of rickety beads; the latter being due mainly to overgrowth of cartilage, and never persisting into adult life.

In three or four other cases I have noticed hypertrophy of the ends of the long bones in acute rheumatism; sometimes the coronoid process of the ulna is felt thickened, sometimes its lower end near the styloid process, as in Case 1; while in the case just related, many bones were affected. There is often great tenderness over the thickened portion of the bone, suggesting periostitis, and sometimes a little fibrous nodule may be felt adherent to the presumably-inflamed periosteum.

With regard to the cases of general muscular atrophy, are we to regard them as examples of a general trophic change set up reflexly by much joint irritation, or have we, as the anæsthesia in one case would seem to indicate, a degeneration of the extremities of a large number of peripheral nerves?

Local palsy and muscular atrophy are also met with in cases of *chorea.*

Case 11.—A female, aged 20, under the care of Dr. Simpson, had articular rheumatism, affecting mainly the joints of the limbs, just before Christmas. In February twitching began. On admission in March chorea was severe and general. A week after admission a tendency to wrist-drop on both sides was noted, and the left ulnar nerve was distinctly thickened. A fortnight later the muscles on the back of the right forearm were greatly atrophied; the interossei, thenar, and hypothenar eminences were also wasted. The accompanying woodcut shows the flattening of the forearm and the grooving on the back of the hand.



Although the girl was generally very thin and wasted, the contrast between the right and left forearm was striking. The wasted muscles gave diminished contraction to faradism, and an increased irritability to galvanism, but I could not satisfy myself as to a qualitative alteration.

In this case we have the following sequence: articular rheumatism, chorea, paralysis, and atrophy of muscles supplied by the musculo-spiral and ulnar nerves; and it is important to note that, so far as could be ascertained, the joints of the upper limbs were never painful or swollen.

Dr. Railton showed a little girl at a meeting of the Manchester Medical Society, who, after an attack of chorea, developed paralysis and wasting of the lower limbs, some of the muscles giving the reaction of degeneration. The knee-jerks were absent, and there were some slight sensory phenomena; and both Dr. Railton and Dr. Ross thought that the symptoms were best explained by a peripheral neuritis.

A few months ago a girl aged 15, came to my out-patient room with symptoms pointing to a neuritis of the right ulnar nerve. It was ascertained that she had rheumatic fever when nine years old; and chorea two years later. During the attack of chorea the numbness and weakness in the territory of the ulnar nerve commenced. The right foot was unduly arched and the great toe had been drawn up for a year; partial anæsthesia was detected over the anterior part of the foot and toes.

A very remarkable case was described by Dr. Bernard, of Londonderry, at the Belfast meeting of the British Medical Association. A child with chorea had wasting of the thenar and hypothenar muscles, and also of the interossei, and one hand presented a "main en griffe" attitude. It is probable that the chorea was attended or preceded by rheumatism, which had led to a neuritis of the ulnar nerve.

At a meeting of the Clinical Society, London, in 1884, Dr. Hadden showed a patient who, after an attack of acute rheumatism, presented trophic changes of the nails and skin of the fingers, together with impaired sensation and wasting of the muscles of the right forearm; and he mentioned two other cases where patches of anæsthesia were present in the arms and legs. In the discussion which followed, my friend Dr. Thomas Barlow described several cases of his own, short notes of which he has very kindly sent me, and as Dr. Barlow had referred to some of them in a previous but unpublished paper on rheumatism, read at Birmingham some years ago, he has the merit, I think, of first drawing attention to the subject. His cases are as follows .—

(1) A man with hyperpyrexial rheumatic fever, who during early convalescence had definite paralysis of one ulnar nerve and anæsthesia for a few days.

(2) Rheumatic fever, with bad pericarditis ; during early convalescence definite paralysis of one ulnar and anæsthesia for a few days; also moderate wasting along the muscles supplied by the ulnar nerve to the hand. In this case there was also wasting of other limb muscles.

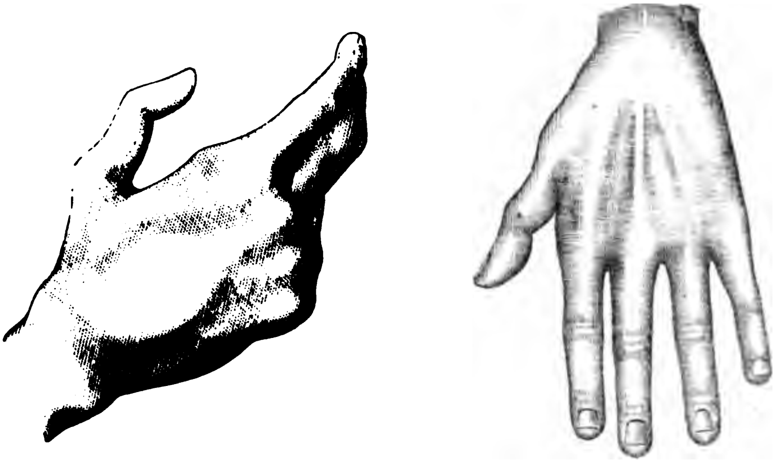
(3) A remarkable case of *extreme* wasting of all the interosseous spaces of the chest, following a very severe attack of rheumatic fever, but without pleurisy to account for the wasting.

(4) Agnes T., in February, 1883, had a slight rheumatic attack, followed in two or three days by chorea, which lasted two months. In November she had another rheumatic attack, followed by chorea, lasting

five months. Again, in August, 1884, she had chorea and intercurrent rheumatism for three months. In January, 1885, æt. eight years, she had chorea, subcutaneous nodules, and a gangliform swelling on the back of the right hand. There was also marked atrophy of the interossei, but no anæsthesia.

(5) Extreme wasting of the deltoid and scapular muscles as sequel to subacute rheumatism associated with a stiff neck.

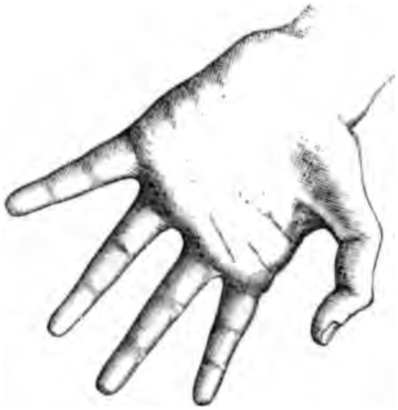
(6) A boy had a slight rheumatic fever and endocarditis; he came to Dr. Barlow some time after with mitral stenosis, stiff neck, and moderate wasting of the muscles of the arm and forearm down the side to which the hand was inclined. There was a slight deficiency in response to faradism, but no other sign of the reaction of degeneration, and no sensory disturbance.



Case (4), Agnes T.

Dr. Fry has reported the case of a girl who, about a month after her third attack of chorea, presented a marked paresis of the lower extremities, which, so far as could be ascertained, had developed within a day or so. The toes dropped in walking; the knee-jerks were gone and the patient complained of tingling in the feet—a few days later she was unable to walk, complained of painful tingling in the feet, legs, and hands, and the muscular power in the hands and forearms was very feeble. Two months after the appearance of the paralysis an electrical examination "showed a reaction of degeneration in the muscles of the hands, feet, forearms, legs, arms and thighs. The muscles were considerably atrophied and already somewhat contractured, the toes being flexed and the ankles extended, the hands and wrists presenting the first stage of the well-described bird-claw deformity. Tactile and temperature sense now almost *nil* over the areas of motor paralysis. The deep and superficial reflexes were gone." Fifteen months later she walked well, but the toes still dropped as she raised her feet in stepping. The hands were nearly normal and muscular power in all the extremities was good. She was last seen about two years after the onset of paralysis and the knee-jerks were still absent. Dr. Fry records the case as one of multiple neuritis, and discusses its etiology. He excludes the influence of arsenic, which the patient had taken in somewhat large doses, previous to the appearance of the paralysis, and after referring to evidence bearing on the infectious origin of some cases of chorea, asks the question, "Was the extensive multiple neuritis, in our case, possibly due to an infectious cause?"

Barbillon, in a thesis on the state of the cutaneous sensibility in acute rheumatism, points out, as Drosdoff did before him, that the faradic sensibility of the skin is usually diminished or abolished in the neighbourhood of the affected joints, and sometimes the diminution may be observed over the whole of an affected limb. Also, that sensibility to touch and pain is often diminished, but to a less degree, and less constantly than the faradic sensibility. He states, too, that muscular weakness and atrophy are frequently associated with the partial anæsthesia, but he only alludes to this incidentally, and does not localise either weakness or anæsthesia to special nerve territories. In discussing the pathogenesis, however, he suggests the possibility in some cases of an affection of the peripheral nervous system.

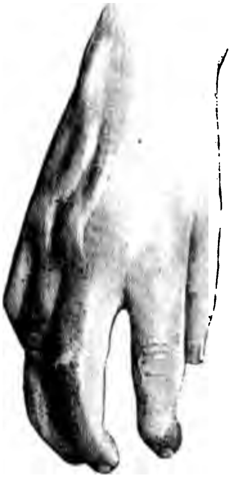


Case (4), Agnes T.

As regards *chronic rheumatic arthritis*, we have the important investigations of Pitres and Vaillard, who examined the peripheral nerves of three cases and found them diseased in all, and there was a close correspondence between the muscular atrophy and the degenerated nerves. Thus, in one case where the leg muscles were atrophied, the corresponding nerves were profoundly altered; in another case, where all the limb muscles were normal in colour and structure, the muscular branches of the nerves were healthy, and the writers suggest a constant connection between the neuritis and the trophic disorders.

Ord has also drawn attention to the muscular atrophy and wasted glossy skin seen in chronic rheumatism, attributed by him, however, to a central cause.

In the hand here pictured the skin over the back of the fingers was thin and glossy, and over the first phalanges showed brownish pigmentation. There was also atrophy of the interossei and thickening of one ulnar nerve.



The gist of the preceding observations appears to be :—

(1) That in articular rheumatism we constantly meet with the muscular atrophy and paresis common to other joint affections, and that while the sudden onset of these symptoms appears to be best explained by a reflex mechanism, whereby irritation conveyed along sensory nerves from the joint to the cord inhibits in some way the functional activity of the motor cells in the anterior horns, the progressive character and duration of the atrophy suggest organic changes either central or peripheral. The presence also of increased tendon reactions, sometimes of contractures, and the fact that very rarely a lateral sclerosis may start from an arthritic attack* indicate that we may have changes not only in the motor cells but also probably in the terminations of the pyramidal

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* Gower's "Diseases of the Nervous System," Vol. I., p. 330.

tract or in that network of nerve fibrillæ by which they become connected with the ganglionic cells, changes possibly not detectable by the microscope, yet, in all probability, more than functional.

(2) That one of the plainest and commonest phenomena of acute, subacute, or chronic rheumatism is wasting of the interosseous muscles of the hand; and that while some cases may be explained in the same way as other arthritic atrophies, viz., reflexly from inflamed knuckles, there is sufficient evidence that in a large number of cases the atrophy is due to an ulnar neuritis. Thus, in ulnar paralysis the index and middle fingers are less affected than the two inner fingers, the outer two humbricales being supplied by the median. Now this is usually, though not invariably, the case in the rheumatic hand (see Fig. page 340), where the little and ring fingers show over-extension of the first phalanges and flexion of the others. In rheumatism too there appears to be a special incidence on the adductor pollicis and the abductor indicis. Confirmatory evidence of neuritis is afforded by the frequency with which impaired cutaneous sensibility is met with in the ulnar nerve territory, along with thickening and tenderness over the trunk of the nerve itself.

(3) That although the ulnar is by far the commonest nerve to be affected, there is substantial proof that other nerves of the brachial plexus and that branches of the lumbar and sacral plexuses are also frequently attacked.

(4) It is of great significance to note that these peripheral nerve symptoms may occur in a limb *quite free from joint irritation*, as, *e.g.*, during early convalescence from rheumatic fever. And if we thus often meet with paralysis, atrophy, or anæsthesia in the course of the ulnar nerve during an attack of rheumatism, or immediately after the pyrexia has subsided, in a limb whose joints are free, it appears to me that we have justifiable if not conclusive grounds for believing not only in a neuritis, but in one set up by the rheumatic poison.

This view, supported by the clinical evidence I have adduced, has been recently confirmed by the careful post-mortem observations of Dr. Gordinier, an American physician. His case was that of a man, aged 31, who had been subject for several years to repeated attacks of rheumatism. He had always been temperate, and denied syphilis. In November, 1888, he was seized with acute articular rheumatism; both ankles were the seat of severe pain on pressure and movement, and were much swollen; next came swelling of the left elbow and shoulder joints, attended by great pain on motion and pressure; shortly afterwards he had severe pain in the cardiac region, and suffered from dyspnœa. When examined, four months later, he was much emaciated, suffered from orthopnœa, and œdema of the feet, and presented well-marked physical signs of mitral stenosis, and later of aortic disease. There was distinct foot and wrist drop. "Decided tenderness of muscles and of nerve trunks in arms and forearms. Deltoids, biceps and extensors of forearms, as well as interossei and muscles of ball of thumb, were markedly wasted. The same tenderness of muscles and nerves existed in lower extremities, particularly in the vasti, muscles of calf, and peronei group, which are atrophic to a marked degree; anæsthesia in spots on outer aspect of ankles; loss of muscular sense in toes, and slight in whole lower extremities; retardation to conduction of pain well marked. Slight anæsthesia in distribution of ulnar nerves; no ataxic movements. Patellar tendon reflexes absent. Bladder and rectum normal." The atrophic muscles presented the reaction of degeneration to electricity. A few days later symptoms of pericarditis appeared, and he rapidly sank.

At the autopsy the nerve sheaths of the ulnar, musculo-spiral, anterior crural, and sciatic nerves were swollen, and full of blood; the vasti, deltoid, and calf muscles were pale and flabby. The heart was much enlarged, and covered with recent exudations and old adhesions; the mitral orifice was narrowed, and the aortic orifice almost occluded with vegetations. The liver presented a nutmeg appearance. Numerous sections were made of the spinal cord and peripheral nerves. The cord was quite healthy. "Sections of the anterior crural and ulnar nerves showed not only an interstitial neuritis but marked parenchymatous degeneration of nerve fibres, such as marked swelling of medullary sheath, with an entire absence of axis cylinders, which were replaced by granular matter; others with segmentation of myeline and beginning granular conditions of axis cylinders, being broken up or segmented, or entirely replaced by granular matter."

(b) PERIPHERAL NEURITIS IN GOUT.

Gouty subjects often suffer from numbness and "tingling pains" in the finger tips, and these symptoms appear to be easily excited by small quantities of alcohol in persons who inherit a strong tendency to gout. Thus, Dr. Ross knew a gentleman of gouty parentage who experienced numbness in the finger tips after taking a single glass of beer, and if he persisted in taking beer to dinner for a few days, his finger-nails became dry, and cracked longitudinally. This susceptibility to alcoholic drinks makes it difficult to decide how far symptoms, indicative of a peripheral neuritis occurring in a gouty subject, are to be attributed to the influence of alcohol or to that of gout. The difficulty is illustrated by an interesting series of cases described by Dr. Buzzard, in which symptoms suggestive of slight neuritis were present, and appeared to depend on gout; but as many of the patients partook pretty freely of alcohol, its influence could not be absolutely excluded.

The symptoms presented by Dr. Buzzard's cases were: Numbness and tingling in the fingers; acute pains radiating along a limb, often especially severe in the thumb or in one finger; cutaneous hyperæsthesia; and Dr. Buzzard mentions one place in particular where exquisite pain was felt on pressure. "It lies just inside the inner and upper angle of the scapula, and the pain caused by the pressure there seems to travel down to the hand. Apparently there is neuritis of the posterior branch of a spinal nerve, the anterior branch of which enters into the formation of the brachial plexus." These sensory phenomena were soon followed by muscular weakness and atrophy, sometimes affecting the greater portion of a limb, but as a rule limited to certain parts, as the intrinsic muscles of the thumb, or some of the forearm muscles.

A patient, under the care of Drs. Ross and Mules, an old gentleman who was the subject of chronic gout in both feet, had almost lost the power of walking. All the muscles of the lower extremities were the subjects of fibrillary contractions, these being found more especially in the extremities of the thighs and legs. These muscles showed also manifest signs of wasting, and the patellar tendon reactions were lost. Under massage treatment the patient gained an inch in the circumference of the calves, and an inch and a half in that of the thighs, and the fibrillary contractions disappeared. The joints of the feet were anchylosed, but the patient regained in great degree his power of walking, although it remained awkward.

Mr. Hutchinson has drawn attention to the occurrence of attacks of neuro-retinitis in gouty subjects; in one of his cases the third cranial nerve was implicated; in another case, the left facial nerve; while another member of a gouty family suffered from neuritis of the brachial plexus.

Cornillon has reported a case which suggests that considerable muscular atrophy may owe its origin to gout. It was that of a man aged 55, who, in the winter of 1880, suffered for a period of ten days from pains in both shoulders and both wrists, the joints being hot and swollen. During the winter of 1881 he had another attack, more painful and longer in duration than the first one. Again the wrists and shoulders were alone implicated. From this time the shoulders became feeble, the forearms wasted, and the fingers crooked. When seen by Cornillon, a few months later, there was marked atrophy of the deltoid, the extensors of the forearms, and the small muscles of the hands. The fingers presented the "main en griffe" attitude. Judging from the presence of gouty tophi in the right ear, Cornillon regarded the arthritic attacks as gouty in nature, and the muscular atrophy in the upper limbs as a sequela of a gouty joint affection. He does not express any opinion as to the cause of the muscular atrophy, but it seems likely that the pathology of this case is that of other arthritic muscular atrophies, a subject already discussed. Hence the question whether the patient suffered from rheumatism or gout is not of much interest or importance.

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(c) Neuritis in connection with the Puerperal State.

The paralyses which occur in connection with pregnancy and the puerperal state are of many varieties. Leaving out of consideration those which depend on disease of the brain or spinal cord, the only ones that call for notice in the present work may be divided into two groups, viz., (1) Paralysis due to injury or disease of nerves within the pelvic cavity. (2) Paralysis due to a multiple neuritis initiated by some condition of the blood or tissues peculiar to the puerperal state.

I.—With regard to the *first group*, the common cause is compression of a nerve trunk by the head of the child, and paralysis is especially liable to occur when delivery has been effected by the forceps; in cases of this kind the paralysis is usually unilateral. Another cause is pelvic inflammation and then the paralysis is often bilateral; the inflammation may spread to the sheaths of the nerves or the nerve trunk may be compressed by an accumulation of pus or strangled by contracting lymph.

Examples of Traumatic Paralysis.—(1) A woman came to my out-patient room with weakness of the right leg. She had had fifteen children, and stated that for three months before her last confinement she was unable to lift up her right leg properly, and that it dragged in walking. The labour was tedious and required the aid of forceps. She felt the forceps slip off the child's head, and screamed with pain. Her right foot was drawn up, she could not move it, and the limb felt numb. She was unable to walk for two months after her confinement. When examined a month later she could voluntarily flex the knee and hip, but was unable to move the foot. The foot was dropped, and the muscles on the front of the leg were slightly wasted. Patches of partial anæsthesia were discovered on the outer aspect of the leg. The knee-jerks were both increased, the right knee-jerk being more exaggerated than the left one; there was no ankle clonus, and the right plantar reflex could not be obtained. Two years later, after undergoing a regular course of massage, the limb had almost completely recovered, but the flexors of the right foot were still weak, and the knee-jerks abnormally irritable.

Vinay describes a case in which there was paralysis of the extensors of the foot, and of the tensor vaginæ femoris, muscles supplied by the external popliteal and superior gluteal nerves respectively. He believes that these symptoms are to be explained by pressure of the child's head on the lumbo-sacral cord (from which the superior gluteal nerve is derived), and on the roots of the sacral nerves.

Paralysis from Pelvic Inflammation.—Leyden has reported the case of a woman who had an attack of puerperal fever immediately after confinement, and became paralysed in the lower extremities, after suffering from neuralgic pains in the territory supplied by the sciatic nerves. At the autopsy a phlegmonous infiltration of the cellular tissue of the pelvis was found, which had implicated the sheaths of both sciatic nerves.

Lever, Bianchi, Imbert-Gourbeyre, and Adams have also described similar cases.

II. *Paralysis from Multiple Neuritis.*—Moebius has recorded seven cases of "puerperal neuritis." He says that in this affection the nerves of the upper limbs, usually the terminal branches of the median or the ulnar nerve are principally involved. One of his patients was a multipara, thirty years of age. Within a month after delivery—which had been entirely uneventful—she began to complain of pain in the hand, and the flexor tendons of the middle finger were stretched like a cord across the palm and were very painful to touch. Both hands became affected in this way, and the patient could no longer use them; a fortnight later she had shooting and pricking sensations in the thighs and buttocks and began to get weak on her legs. The hands were the only parts that were obviously atrophied, and especially in the first interosseous spaces. There was no anæsthesia, and the tendon reactions were obtainable in the upper extremity. The patient was able to get about, but was readily fatigued. There was also paræsthesia of the hands and feet. Some months later recovery was almost perfect, but the patient still complained of lancinating pains in the arms. The atrophy of the interossei completely disappeared.

Dr. Strain relates the case of a woman æt. 32, who, on the second day after her sixth confinement, which was unattended by any difficulty, found that her left leg was powerless from the knee down. On the next day she complained of loss of power in both legs, accompanied by a "prinkling" sensation from her knees down to her toes. There was marked paralysis of the flexors of the feet. Plantar reflexes and ankle clonus could not be obtained, but the knee-jerk could be feebly got in both legs. Sensibility was normal except on the dorsum of the foot, where light impressions were not readily felt. Both legs were very œdematous. Micturition and defæcation were normal; there was no pyrexia; milk had come and the lochial discharge was natural.

At the end of the week the œdema had extended to the thighs. The knee-jerks were absent, and, with the exception of being able to flex the thigh, she was almost completely paraplegic. About this time she began to complain of a "prinkling" in her hands, which were found to be œdematous as far as the wrists, and decidedly weak. The cutaneous sensibility was diminished in both hands and feet. A little later a change in the tone of her voice was observed, and she became very anæmic. Her condition for some time remained stationary, but after a period of about three months the first symptoms of improvement appeared, which consisted in the gradual return of power in the extensors of the hand, together with disappearance of œdema and prinkling. Subsequently her improvement was slow but sure, "and it was curious to to note how with return of power disappearance of dropsy and prinkling took place." The muscular atrophy was not greater than could be accounted for by so many months of disuse. "At the end of a year she was practically well, but informed me that at certain times she still felt her feet sleepy, and that on these occasions there was always a puffiness about her feet and ankles."

Dr. Strain is of opinion that the paralysis depended on a multiple peripheral neuritis, and after excluding alcohol, syphilis, beriberi, and other causes of this disease, he is inclined to regard the marked anæmia as an important ætiological factor of the case.

A patient under the care of Dr. Whitfield suffered from severe vomiting during the whole of her pregnancy. A fortnight before confinement she felt her legs cold and found that she was losing the use of them, and she had to be assisted up and down stairs. The labour was quite natural, and vomiting ceased on the following day. After the fourth day she complained of numbness in the legs, and in a few days of pains in them, of "pins and needles" in the hands and a burning sensation in the palms, as well as of pains up the arms. She could move her legs slightly in bed, and her wrists did not drop till the twelfth day after confinement. On the thirteenth day Dr. Dreschfeld saw her in consultation with Dr. Whitfield. At this time the legs were very weak and the reflexes absent. Both feet were extended and the toes flexed, the hands were dropped, and power to extend the wrist and fingers was lost. The patient had difficulty in raising herself in bed, and complained of a numb feeling round the lower part of her abdomen. Cutaneous sensibility varied in different parts, being increased in some places and diminished in others. Pain was caused by pressure on the main nerve trunks of the upper and lower extremities. The patient ultimately recovered, but very slowly. She had always been strictly temperate.

Dr. Handford relates the following cases:—(1) A woman, aged 43. No evidence of drinking habits, but her husband keeps a public-house. Three weeks after her fourth confinement she lost power in the legs completely, and felt strange sensations like "pins and needles" in the arms. This was soon followed by paralysis. The general character and course of the paralysis was typical of an alcoholic multiple neuritis. There was loss of muscular sense, diminished cutaneous sensibility, but greatly increased deep sensibility, general muscular weakness, loss of the knee-jerks, and the reaction of degeneration in the muscles of the arms and legs. She was able to walk a little in six months, and in twelve months was completely well.

(2) The wife of a public-house keeper, immediately after her confinement, was seized with complete paralysis of the extensors of the toes and the flexors of the ankles, and with weakness of most of the other muscles of the legs. There was a little œdematous swelling and considerable hyperæsthesia, both superficial and deep. The muscles below the knees gave no response to a strong faradic current nor to galvanism. Some weeks later there was much muscular wasting, which extended as high as the right gluteal muscles. The patient could walk in six months, and eventually recovered.

Dr. Handford considers that the multiple neuritis in these cases was probably of alcoholic origin, the onset being determined by child birth. He does not regard child bearing an efficient cause *per se* of multiple neuritis, but suggests that the lowered condition of "tissue health" during the puerperal period predisposes to neuritis; and he asks, "Is the influence of child bearing one of the factors which renders alcoholic neuritis so much more common in women than in men, while drinking habits are more prevalent in the latter?"*

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* For other considerations relating to the frequency of alcoholic paralysis in men and women, see page 151.

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Korsakow und **Serbski**. "Ein Fall von polyneuritischer Psychose mit Autopsie."—*Archiv für Psych.*, Band XXIII., Heft 1, N.C. 1891, p. 471. Symptoms of multiple neuritis followed laparotomy for the removal of an extra-uterine fœtation; degenerative neuritis of a large number of spinal nerves was found, and was probably of septic origin.

(*d*) ON THE KNEE-JERKS AND PERIPHERAL NEURITIS IN DIABETES.

THE fact that the knee-jerks are frequently absent in cases of diabetes mellitus was pointed out by Bouchard[1] in 1884. The absence of knee-jerks is now a well known symptom in the disease, and there are many points of interest in connection with this peculiar reflex. From my notes of cases of diabetes seen at the Manchester Royal Infirmary and met with elsewhere, I have selected fifty, and the following are a few statistics respecting the knee-jerks in these cases.

Proportion of Cases having the Knee-jerks Absent.—In fifty cases the condition of the knee-jerks was as follows:—

Knee-jerks absent	in 25 cases	= 50 per cent.
One knee-jerk absent, one obtained feebly (Jendrassik's method)	in 3 „	= 6 „
Both knee-jerks just obtained, but very feeble (Jendrassik's method)	in 3 „	= 6 „
Knee-jerks present	in 19 „	= 38 „
	50	

If the knee-jerks were not obtained in the ordinary manner Jendrassik's method or that described by Buzzard[7]* was employed; otherwise the proportion of cases of absent knee-jerks would have been still higher, as in some cases the knee-jerks were not obtained in the ordinary manner, but were elicited by these methods. In the above table the proportion of cases of absent knee-jerks is higher than in statistics hitherto published. Thus Bouchard[2] found the knee-jerk absent in 28 per cent of his cases, and in a later series in 36·9 per cent; Auerbach[3] in 35-40 per cent; Maschka[4] in about 30·6 per cent; very feeble in 19·4 per cent. Eichhorst[5] found the knee-jerk absent in 9 out of 48 cases = 20·9 per cent. He points out the difference between private and hospital cases. In 36 private patients the knee-jerks were absent in 6 = 16·7 per cent; in 7 hospital patients they were absent in 3 = 42·9 per cent. Eichhorst states that his hospital cases were suffering from a more severe form of the disease than his private cases, and this may be the cause of the difference. It may be mentioned that nearly all the cases in my table were hospital patients, mostly suffering from a severe form of the disease with wasting, and very few were cases of the diabetes of stout old people. This may be the reason why the percentage of cases, in which the knee-jerks were absent, is higher than in previous statistics.

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* The patient is seated on a chair, with his feet well in contact with the floor, and the knees bent at an angle just a little more than a right angle. He is made to look upwards, to link his fingers together, and to pull tightly. The observer places one hand on the rectus femoris muscle and strikes the patella ligament with a percussion hammer or stethoscope. If the slightest knee-jerk be present, a contraction of the rectus muscle will be felt.

Variability of the Reflex.—A knee-jerk, which has disappeared in a patient suffering from diabetes, may return again and vary very much in the course of time. Thus in a case, J. L., aged 36, in September, 1890, the knee-jerks were *absent*, urine daily quantity 210 to 280 ozs., sp. gr. 1030, acid, no albumen, sugar = 27 to 30 gr. to the oz.; November 25, knee-jerks *present*, urine sp. gr. 1032, 26 gr. sugar to oz., trace of albumen, marked reaction with Fe_2Cl_6; June 30, 1891, knee-jerks *absent;* September 17, 1891, patient improved, right knee-jerk *present* but very feeble, left knee-jerk *absent.*

In another case, J. D., aged 52 (urine 1030, 26gr. of sugar to oz., amount of urine 150—170 oz. daily), the knee-jerks were absent (Jendrassik's method) May, 1892. June 29th, knee-jerks present; July 20th, knee-jerks well marked.

Knee-jerks which are present when the patient first comes under observation, may, of course, be lost at a later period in the disease. But this is certainly *not* always the case, as the knee-jerks may be obtained after the disease has existed for a long time, and even up to the death of the patient. I have found them present at the end of 2¼ years, and, in another case, they were present, but feeble, at the end of 5 years: also in three cases that terminated fatally in diabetic coma, the knee-jerks were present up to the very last.

Relation to Age.—The condition of the knee-jerks in diabetes is influenced by the age of the patient. The following table shows clearly that the knee-jerks are lost in a greater proportion of cases under the age of thirty than in cases over thirty.

In forty-two cases of diabetes:—

Between the ages of	Knee-jerks absent in	Knee-jerks present in	
10 and 20 years	4	1	∴ Under the age of 25, knee-jerks absent in 8 out of 10 cases = 80 per cent.
20 and 25 years	4	1	
25 and 30 years	4	2	
	—	—	∴ Under the age of 30, knee-jerks absent in 12 out of 16 cases = 75 per cent.
	12	4	
30 and 40 years	4	5	
40 and 50 years	2	5	
50 and 60 years	5	2	
60 and 70 years	1	2	∴ Over the age of 30, knee-jerks absent in 12 out of 26 cases = 46·15 per cent.
	—	—	
	12	14	

It is well known, that in young persons, diabetes is of a much more severe type than in middle or advanced life, and this may be the reason why the knee-jerks are absent in a greater proportion of cases under the age of 30.

The absence of knee-jerks does not appear to bear any definite relation to the *duration of the disease.*

In two cases they were absent when the patients were examined five weeks after the first onset of diabetic symptoms, whilst in another case (with wasting) the knee-jerks were present three years after the first onset of symptoms. In 32 cases examined during the first twelve months after symptoms of diabetes were noticed by the patient, the knee-jerks were absent in 17 cases = 53·1 per cent. In 11 cases examined after the first twelve months, the knee-jerks were absent in 7 = 63·6 per cent.

Relation to General Nutrition.—As most of the cases examined were severe forms of the disease, with wasting, they did not form a very suitable series for comparison of the condition of the reflexes in stout and wasted patients. Only a few were cases of the milder forms of the disease occurring in well-nourished elderly people. Still the knee-jerks appear to be more frequently absent in markedly wasted patients than in those who are fairly well nourished. In 15 patients markedly wasted the knee-jerks were absent in 10 = 66·6 per cent, very feeble in 2, present in 3. In 7 patients wasted, but to a less extent, the knee-jerks were absent in 2, very feeble in 2, present in 3. In 8 cases in which the general condition was good the knee-jerks were present in 6; in 2 cases one knee-jerk was absent and one present, but very feeble. Hence, in 15 cases markedly wasted, they were absent in 66 per cent, whilst in 8 patients, whose general condition was good, in no case were both knee-jerks absent.

Relation to the Perchloride of Iron Reaction in the Urine (Gerhardt's reaction).—The absence of knee-jerks did not appear to bear any relation to the brownish-red colouration, so frequently obtained in diabetic urine by the addition of perchloride of iron (so-called reaction for diacetic acid). Thus in 20 patients whose knee-jerks were absent, the reaction with perchloride of iron was present in 11, very slight in 1, absent in 8. In 15 patients whose knee-jerks were present, the perchloride of iron reaction was obtained in 7, very slight in 3, absent in 5.

Relation to Pathological Conditions.—The loss of the tendon reflex cannot be regarded as evidence of the nervous origin of the disease in any particular case, but must be looked upon simply as a complication. In a case of diabetes presenting symptoms of gross lesion of the nervous system, the knee-jerks were present.*

In another case of mild diabetes, in which a tumour of the pituitary body was found post mortem, the knee-jerks were present. On the other hand, they were absent in a case following a blow on the head, and in cases having a previous history of great mental worry. The knee-jerks were absent in three cases in which the autopsy revealed marked disease of the pancreas.

Relation to the Condition of the Urine.—The cases examined did not show any definite relation between the loss of knee-jerks and the quantity and specific gravity of the urine, or the amount of sugar present. In the few mild cases the knee-jerks were present, but in the other more severe forms of the disease (the majority of the cases) there was no relation between the knee-jerks and the condition of the urine.

Rosenstein[6] is of opinion that the loss of knee-jerks stands in no relation to the amount of sugar in the urine, nor to the presence of acetone or the perchloride of iron reaction. He found the knee-jerks absent in two mild cases in which the sugar in the urine was only 0·7 per cent and 0·5 per cent respectively. He also believes that the loss of the knee-jerk has no relation to the general nutrition or to bodily weakness. Subcutaneous injection of strychnia did not cause the knee-jerks to return.

Relation of the Absent Knee-jerks to Symptoms of Neuritis.—In twelve cases that had lost the knee-jerks, there were in eight more or less pain, tenderness, numbness, and cramps in the legs—symptoms which are characteristic in the early stages of alcoholic peripheral neuritis. In four cases such symptoms were entirely absent. There were also marked paralytic symptoms in two of the eight cases above mentioned. Of ten patients whose knee-jerks were present, only one suffered from the above symptoms. In this case there was slight numbness, tingling, and tenderness in the legs. Hence pain, tenderness, numbness, tingling, etc. (which may be considered early neuritic symptoms) were present more frequently in patients whose knee-jerks were absent, than in those whose knee-jerks were present. Still in many cases the knee-jerks are absent, and yet the patient does not suffer from any of the above-mentioned symptoms of early neuritis.

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* See *Lancet*, July 9th, 1892.

When the patient suffers from symptoms such as pains, numbness, tingling, cramps, tenderness, etc., in the legs, the loss of knee-jerks may be attributed, and probably rightly, to peripheral neuritis. But the cause of the absence of knee-jerks is not so evident in cases where the above-mentioned symptoms are absent. Are the absent knee-jerks in these cases to be considered as the first sign of a commencing neuritis, or is there some other cause?

Buzzard,[9] in 1886, attributed the loss of knee-jerk, in some cases to peripheral neuritis, but states that he does not know whether this is always the cause. Charcot believes that peripheral neuritis is probably the explanation of this sign.

As regards the pathological evidence, it may be mentioned that the spinal cord has been examined microscopically (in cases in which the knee-jerks were absent during life) by Rosenstein,[6] Nivière, Levia, Nonne, and others. In all cases it was normal. In Nonne's case* not only the spinal cord but also the peripheral nerves (anterior crural) were normal. Some time ago I examined the peripheral nerves in a case of severe diabetes with wasting. The knee-jerks were absent, but there were no other signs of peripheral neuritis during life. The anterior tibial nerves and their branches were normal on microscopical examination. The branches of the anterior crural nerve were unfortunately not examined.

Eichhorst[8] has recently reported two cases of diabetes with absent knee-jerks, in which he found parenchymatous neuritis of the anterior crural nerves. In patients whose knee-jerks are absent, but who have no other motor or sensory symptoms, he attributes the loss of the reflex, in some cases, to peripheral neuritis; in other cases, as in that reported by Nonne, where the peripheral nerves were normal, to a functional disturbance, probably of toxic origin. He thinks it is probable that this functional disturbance precedes peripheral neuritis. In those cases in which the knee-jerks are lost, but within a short time return, and vary with the severity of the patient's symptoms, he believes the cause of the disappearance of the reflex to be a functional disturbance. When the loss of knee-jerks is due to neuritis, then a much longer time will elapse before they return (if such an event should ever occur).

The positive pathological evidence gives an explanation of the loss of knee-jerks in some cases. Still, in other cases, the clinical facts, that the condition of the knee-jerk varies so considerably—at one time absent, then soon afterwards present, and perhaps again lost at a later date—and that no other symptoms of neuritis may be present, support the view of a toxic functional cause (probably a condition preceding neuritis) in these cases.

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* Quoted by Eichhorst.[8]

Prognosis.—Whilst some writers do not attach any prognostic value to the loss of knee-jerks, others consider that the prognosis is distinctly more unfavourable in cases in which the knee-jerks are absent. Bouchard, Nivière, Marie, and Guinon[2] hold the latter view, and give statistics in favour thereof. As above mentioned, the knee-jerks in some cases persist to the last; also, they are sometimes absent in mild cases. The following figures, however, are in favour of the view that the prognosis is generally more unfavourable when the knee-jerks are absent. Thus, from my notes of cases of diabetes in Manchester I find that:—Of 25 patients whose knee-jerks were absent on admission into hospital, 7 died whilst under treatment in the hospital (*i.e.*, within 6 weeks of admission) = 28 per cent; of 18 patients whose knee-jerks were present on admission, only 1 died whilst under treatment in the hospital = 5·5 per cent.

I have ascertained the after-history of 20 diabetic cases in Manchester, and the following table shows the duration of life in 10 patients whose knee-jerks were absent, and in 10 patients whose knee-jerks were present, on first coming under observation:—

DURATION OF LIFE FROM DATE ON WHICH THE CONDITION OF THE KNEE-JERKS WAS FIRST ASCERTAINED.

	When patient first came under observation	
	Knee-jerks absent in 10 cases.	Knee-jerks present in 10 cases.
Died within 2 weeks	4	1
„ between 2 weeks and 6 months	2	3
„ „ 6 months and 12 months	2	1
∴ died within 12 months	8	5
Died between 1 and 2 years	1	3
Alive 2 years afterwards	1	2
∴ survived over 12 months	2	5

Hence, of the patients whose knee-jerks were present when first examined, 50 per cent lived over 12 months, whilst only 20 per cent of those whose knee-jerks were absent lived over this period; *i.e.*, 50 per cent of the former, but 80 per cent of the latter cases, died within 12 months.

I have ascertained the duration of life from the first onset of diabetic symptoms to the time of death or up to the present date, in 10 patients whose knee-jerks were absent when first examined. I find that the *total* duration of the disease was under 2 years in 9 of these cases [under 6 months in 2; 6 to 12 months in 3; 12 to 24 months in 4.] One patient was alive at the end of 2 years.

The duration of disease (from first onset of diabetic symptoms to the time of death or to the present date) in 10 patients whose knee-jerks were present when first examined, was under 2 years in 5 cases [under 6 months in 1; 6 to 12 months, 2 cases; 12 to 24 months, 2 cases]. In 5 cases the duration was over 2 years; (in one case 2 to 3 years; in 2 cases 4 years; and in 2 cases the patients were alive and improved somewhat in the third year of the disease).

Hence, of the 10 patients whose knee-jerks were absent when first examined, the *total* duration of the disease was over 2 years in 1 only = 10 per cent; whilst of 10 patients whose knee-jerks were present, the *total* duration exceeded 2 years in 5 cases = 50 per cent.

Many things influence the course of the disease in diabetes, and it is somewhat difficult to come to a definite conclusion as to the prognostic value of a single symptom. Still the above statistics point to the absence of knee-jerks as a sign rendering the prognosis decidedly more unfavourable.

Peripheral Neuritis in Diabetes.—Whilst some of the nervous affections occurring in diabetes are to be attributed directly to the diabetic condition, others, such as hemiplegia from cerebral hæmorrhage or softening, are accidental complications, or are not directly dependent on the disease. The occasional occurrence of various degrees of paralysis and the frequency of neuralgic pain in the limbs have been mentioned by many of the older medical writers, but it is only within recent years that any of these symptoms have been attributed to peripheral neuritis.

Some of the nervous symptoms met with in diabetic patients strongly resemble those of alcoholic and other forms of peripheral neuritis.

v. Ziemssen[8] was the first, in 1885, to attribute the neuralgia so often observed in diabetes to peripheral neuritis. Soon afterwards v. Hoesslin, and at a later date, Eichhorst supported this view. Pryce[10] has reported a case of diabetes with ataxic symptoms, in which the peripheral nerves showed evidences of neuritis. Leyden,[11] Althaus,[12] Charcot,[13] Buzzard,[14] Auché,[15] Burns,[16] have reported cases of peripheral neuritis in diabetes, or, to be more exact, cases presenting symptoms similar to those of neuritis from alcohol or other causes.

The proportion of cases of diabetes that suffer from marked symptoms of peripheral neuritis is very small. But cases presenting slight symptoms are common.

The following are brief notes of two cases presenting slight symptoms of peripheral neuritis, such as are frequently met with :—

(1) T. R., aged 26. Urine 1040; no albumen; loaded with sugar. When patient first came under my care, the knee-jerks were present; there were frequent cramps in the legs. Ten months later patient complained that he had to drag his legs in walking. There was slight tenderness of the calf muscles and frequent cramps; he complained of great pain, of a gnawing character, in the calf muscles, and in the front of the thighs; also he had pain over the tibiæ. Left knee-jerk absent; right, very feeble (Jendrassik's method). At a later date he also complained of tingling and numbness in the legs. No real paralysis, no anæsthesia.

(2) J. R., aged 47. When the patient first came under my care the knee-jerks were absent, but there were no other signs of neuritis. Urine 1032, pale, acid, no albumen, contained a large amount of sugar. At a later date both legs became numb; there was also tingling and pain in the legs, and the calf muscles were tender. The legs became *so tender that patient "could not bear one leg on the top of the other in bed."* Patient could feel and localise touch with a pin's head, and could distinguish between the point and the head of a pin. Movements of legs weak, but no real paralysis.

In both of these cases the symptoms resemble those met with in the early stages of alcoholic peripheral neuritis. In neither case was there a history of alcohol, nor anything to which the symptoms could be attributed except diabetes.

When House Physician to Dr. Buzzard at the National Hospital for the Paralysed and Epileptic, Queen Square, London, I had the opportunity of observing a well-marked case of diabetic neuritis. Dr. Buzzard has since recorded the case in the *British Medical Journal*, and as it is one of the most marked on record, I add a brief abstract, taken from the report there published :—

J. K., æt. 55. Ten months before the patient came under Dr. Buzzard's observation, he began to suffer from severe pain and tenderness on the front of the right thigh, which soon extended down to the foot. A month later the left leg was affected in the same way. Both legs became weak, and at the end of three months he was unable to walk. He then began to suffer from "pins and needles" in the soles of the feet, and at a later date had numbness and tingling in the tips of the fingers. No affection of bladder. Soon after the onset of illness, thirst, diuresis, and wasting became prominent symptoms.

During the time the patient was under treatment the chief symptoms were:—Loss of power in the legs; inability to dorsiflex the feet; dropped toes of left foot; wasting of muscles; reaction of degeneration in anterior tibial muscles; absence of knee-jerks and plantar reflexes;

continuous pains in the legs and feet; tenderness of the legs and soles of the feet; numbness and tingling in the legs and diminished cutaneous sensibility; pains in the fingers, and diminution of tactile sensation in the hands. The bladder was not affected. On the outer side of the right foot, just below the malleolus, was a deep ulcer, surrounded by an area of congestion, and there was also a cicatrix of an old ulcer just below the metatarso-phalangeal joint of great toe.

Urine: 57 to 84 ounces daily; sp. gr. 1042 to 1045. Sugar, 25—35 grains per ounce, according to diet. By rigid diet the sugar was reduced to 16 grains to the ounce; the ulcers healed, and patient improved.

Through the kindness of Mr. Milner, Surgeon to the Salford Union Hospital, I have recently had the opportunity of observing a diabetic patient under his care, who presented marked paresis of the arms. The following are my notes of the case:—

J. D., aged 52.—*Paresis and wasting of right deltoid, pectorals, biceps and triceps muscles.*—On left side the same muscles affected, but to a less extent. Previous health good until nine months ago, when he suffered from a severe cold. Whilst recovering he began to be troubled with great thirst. No family history of diabetes. No history of injury or alcoholism. Six months ago the left arm became weak. At that time no weakness of right arm had been noticed. Three months later the patient regained power in the left arm, but at this time the right arm became weak, and has continued weaker than the left up to the present time.

May 30th, 1892.—Patient is much wasted. Urine 1040, no albumen, contains a large amount of sugar, 20—26 gr. to the oz., quantity of urine 140—170 oz. daily. Arteries atheromatous, radials calcareous. No affection of heart or lungs can be detected. The patient cannot raise the hands to the mouth, but can flex at the elbows and perform movements of fingers. Abduction and other movements at shoulders very feeble. Slight foot drop. Patient can dorsiflex feet, though feebly. Knee-jerks both absent. Patient complains of numbness of the legs, and frequent cramp in the calves of the legs at night.

June 29.—Patient is able to walk, but drags his legs and walks with his feet widely apart. No ataxia. Slight dropping of the foot. When in bed he can raise his legs in the extended position from the bed quite well. The thigh muscles are in a fairly good state of nutrition. Slight œdema of the feet. Both knee-jerks are now present, but are very feeble. Frequent cramps in the calf muscles at night. No other sensory symptoms in the legs. Wasting of biceps, triceps, deltoid, pectorals, and scapular muscles of each side, especially the right. The forearms and hands are only very slightly wasted, and present a well marked contrast to the shoulder and upper arm muscles. The posterior borders and lower angles of the scapulæ project, especially the left. This projection is more marked when the arms are raised. When the patient shrugs his shoulders the upper part of each trapezius is felt to contract. Patient has difficulty in placing right hand on left shoulder, but can place left hand on right shoulder fairly well. The clavicular part of the right pectoral is markedly wasted, and gives rise to a deep depression below the clavicle. When the hands are clasped together firmly between the knees, the left pectoral muscles become much more prominent than the right. Patient is only able to abduct at the shoulder to a slight extent; he cannot raise the arms into a horizontal position—not more than to an angle of 45° (with the trunk). Flexion at the shoulder is much feebler on the right side than the left; the arm cannot be brought into horizontal position; it can only be raised to about an angle of 45°. The left arm can be flexed at the shoulder into the horizontal position. When the arms are raised and flexed in front of the chest there is pronation of the forearms and some flexion at the elbows. Patient can extend the shoulders fairly well on both sides, and he is able to place each hand on the sacrum. Extension of both elbows feeble; flexion at the right elbow very feeble; flexion at the left elbow somewhat feeble, but much better than on the right side, and much better than extension at the same joint. Patient has great difficulty in raising the right hand to his mouth. He is obliged to feed himself with the left hand. He cannot raise a pot of water to his mouth with the right hand, but is able to do so with the left. (Patient was a right-handed man). He complains of a cold feeling in the fingers and hands, also of numbness and tingling in the fingers. He complains of having no feeling in the fingers (right hand especially). He has often to place them in warm water to "get the feeling back." The fingers are swollen, especially the terminal phalanges, which are bulbous, but the skin is pale. Patient is able to feel slight tactile sensations and to distinguish between the head and point of a pin on each arm and hand.

July 20.—Knee-jerks now normal, no ankle clonus, right plantar reflex present, left absent. Abdominal epigastric and cremasteric reflexes normal. Patient is able to feel and localise touch with a pin's head, and to distinguish between the head and point of a pin quite well all over the arms and legs. Flexion at the right elbow exceedingly feeble, and only the slightest prominence of the belly of the supinator longus can be felt when the elbow is flexed, the forearm being midway between pronation and supination. The left supinator longus is felt readily when same movement performed. Right biceps and supinator longus greatly wasted.

From the absence of any cerebral symptoms and the absence of symptoms pointing definitely to lesion of the cord, the previous marked affection of the left arm followed by partial recovery, the presence of numbness, etc., in the fingers, it appears probable that the case was one of slight peripheral neuritis, in which the motor symptoms were most prominent. Or if not the result of an actual neuritis, the symptoms may have been due to a functional affection of the peripheral nerves, caused by some toxic products—a condition preceding neuritis.

Lasèque and Charcot* have described a number of cases of monoplegia and paralysis of single groups of muscles in diabetes. Lecorche points out the rarity of these cases, and states that they do not appear to be connected with any profound lesion of the nervous system: that they are characterised by the paralysis being incomplete, localised, and transitory, and generally sensory symptoms, hyperæsthesia, or anæsthesia, are present. It seems probable that these cases are due to an affection of the peripheral nerves.

In the case J. D., above recorded, the swollen condition of the ends of the fingers with coldness, numbness, and tingling was noted.

In another case (under the care of Dr. Leech) this condition was more marked.

J. L., aged 36, admitted into the Manchester Royal Infirmary, Sept., 1890. Marked diabetes with wasting; knee-jerks absent. After the patient had been in the Infirmary for several months, and about eleven months from onset of diabetic symptoms, the palms of the hands and tips of the fingers became red and flushed. The ends of the fingers and thumbs of both hands became very much swollen and bulbous. The terminal phalanges only were affected, and especially the palmar surface. The terminal phalanges were flushed and of a bright red colour, especially on the dorsal surface. The redness extended as far as the last phalangeal joint. It was most marked on the dorsal surface, and disappeared on pressure. The patient complained of a burning sensation in the ends of the fingers, especially after washing his hands. He also had a throbbing aching sensation in his fingers. At a later date the toes became slightly affected in a similar way.

In the absence of microscopical examination of the nerves of the affected part, of course the pathology cannot be definitely settled. Still it seems probable that in both cases the condition was a vasomotor affection, due to peripheral neuritis.

Résumé of Symptoms in Diabetic Neuritis.—On an analysis of 16 cases recorded by various authors during the last four years, the following description of symptoms is based. Slight symptoms of neuritis, such as pain in the legs, cramps, numbness, tingling, tenderness, and absence of knee-jerks are not infrequent. Cases of diabetes presenting marked paresis or paralysis appear to be comparatively rare. The onset of symptoms is gradual or sub-acute.

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* Quoted by Lecorche[20] and Bernard and Féré.[21]

Motor Symptoms.—Paresis or paralysis, when present, most frequently affects the legs—neuritic paraplegia (Buzzard) or diabetic paraplegia. In some cases (Buzzard[14] and Charcot[13]) the anterior tibial muscles are chiefly affected. There is dropping of the toes and feet, and the patient is unable to dorsiflex the feet. In other cases (as in three recorded by Burns) the paralysis affects chiefly the muscles on the front of the thighs, supplied by the anterior crural and obturator nerves, and the symptoms are sometimes much more marked on one side than the other. In a case recorded by Auché these muscles were affected on one side only, and the patient was unable to go upstairs. In some cases (Leyden,[11] Salomonsen[21]) the motor symptoms are described simply as weakness in the legs. The arms may be affected without the presence of any paralysis in the legs. One arm only may be paralysed, or one arm may be affected at first, but at a later date both may be affected (Buzzard).[14] In other cases the muscles of the shoulder and upper arm may be affected on one or both sides; or the paralysis may be localised to a group of muscles, as the muscles supplied by the ulnar nerve (Ziemssen), or to a single muscle, as the deltoid (Althaus).[12]

The affected muscles are generally wasted. In one case (Charcot) they are said not to have been wasted. The knee-jerks are absent when the legs are affected. Diminished excitability to electricity, and partial or complete reaction of degeneration have been often observed in the affected muscles.

The sensory symptoms are often more marked than the motor, and may be present when the motor symptoms are very slight or absent. When motor symptoms are present, the sensory are generally localised in the same region; in the legs below the knees, where the anterior tibials are chiefly affected; in the front of the thighs where the muscles of this region are chiefly affected (as in Burns' cases); in the arm (Buzzard), or in the region of the ulnar nerve (Ziemssen), or circumflex nerve (Althaus). The hands and feet are often affected. In some cases in which the legs are chiefly affected, sensory symptoms are also present in the hands, and the grasp is weak.

The sensory symptoms are: Pain, neuralgic in character, often described as intense, violent, shooting or tearing; hyperæsthesia, tenderness and pain on pressure; tingling, numbness; sometimes a sensation of coldness in the hands and feet. Diminished tactile sensation is rare, but even anæsthesia may occur (Buzzard's[14] cases); also diminished sensation to pain is recorded. Neuralgia often occurs in diabetes without motor symptoms, and is characterised, according to Berger, by its spontaneous origin, frequent localisation in the branches of the sciatic nerve, sural and plantar nerves, by the violence and long duration of the attacks, by the occurrence of vaso-motor disturbances in the district of the affected nerves, and by its resistance to ordinary treatment for neuralgia, but by its improvement under anti-diabetic treatment. The cause of neuralgia, at least in a certain number of cases, is probably neuritis. In some cases the nerves affected have been noted to be tender to pressure.

The condition of bladder and rectum has frequently not been stated, and therefore probably it was normal. In other cases it was stated to be normal. In one case (Charcot) there was slight incontinence of urine.

Ulcers on the feet,[14] shining and glossy skin, ecchymoses, shedding of the nails, and œdema have been recorded. Herpes zoster has also been recorded.[23] Ataxia has been occasionally noted.

Leyden recognises three forms of peripheral neuritis in diabetes:—

(1) The *hyperæsthetic or neuralgic* variety, in which there is more or less severe pain. This variety may occur in the form of neuralgia (trigeminal neuralgia, sciatica, etc.) or as a multiple neuritis in the feet legs, and hands. Usually there is weakness of the affected parts. Pain stands out as the prominent symptom.

(2) The *motor or paralytic* form. In this form there is more or less marked paralysis of muscles of the legs or of other muscular groups. The knee-jerks are lost. Electrical changes are sometimes found. Often in this form there are neuritic pains.

(3) The *ataxic* form, so-called pseudo-tabes. In this variety, besides ataxia, there are sensory symptoms, numbness, formications in the feet. The muscular power for coarse movements is maintained, or not essentially diminished. The tendon reflexes are lost. The pupils react to light and accommodation, and in this respect, therefore, the cases differ from many cases of true locomotor ataxia. Some neurologists, however, doubt the occurrence of real ataxia in diabetes.

As Charcot points out, however, occasionally, though *very rarely*, a patient may present symptoms of true tabes dorsalis with diabetes. He recognises two groups of possible cases. (1) In cases of tabes dorsalis, symptoms of diabetes may occur, the latter being a complication, and due to the extension of the lesion to floor of the fourth ventricle. In these cases gastric and laryngeal crises are often observed. Marie and Guinon examined the urine in 50 cases of tabes without finding glycosuria in a single case. Gillas found glycosuria three times only in 100 cases of tabes. (2) There is the possibility of the occurrence of tabes and diabetes in the same patient as a coincidence.

Pathological Evidence.—In most of the cases of peripheral neuritis recorded, the diagnosis rests on clinical evidence—the similarity of the symptoms to those of neuritis produced by other causes; the absence of any symptoms definitely referable to the spinal cord or brain, and the absence of any of the other recognised causes of neuritis, such as alcohol, lead, diphtheria, etc.

In a few cases the diagnosis has been confirmed by post-mortem examination—in 1 case reported by Pryce, in 2 cases reported by Eichhorst, and in 3 reported by Auché. In all these cases microscopical examination revealed the existence of parenchymatous neuritis in the nerves of the affected parts.

In one of Auché's cases there was paresis of the legs, muscular cramps, and gangrene of the right foot. In another case itching, tingling, and pricking pains in the feet, legs, and hands. Knee-jerks absent. In a third case violent cramps in the calves at night, slight diminished sensation to a pin prick on the dorsal surface of the forearms, subunguinal hæmorrhages, shedding of the nails.

In one of Eichhorst's cases the knee-jerks were absent, but there was no paralysis and no disturbance of the sensation. In the second case the knee-jerks were absent; the patient was able to move the arms and legs, but there was marked muscular weakness of the limbs. Examination of the anterior crural nerves in both cases revealed parenchymatous neuritis.

In Pryce's case there were perforating ulcers of both feet: diminished cutaneous sensibility of both feet and also of the lower thirds of both legs. The knee-jerks were absent. It is also stated that the patient had ataxic symptoms. The examination of the peripheral nerves (by Mr. Bowlby) revealed parenchymatous neuritis, but the ganglion cells of the lumbar region of the cord were atrophied also.

In most of the cases recorded the neuritis has occurred in patients over the age of 50 (in 12 of 16 cases).

All writers on the subject agree that the neuritis does not bear any relation to the amount of sugar in the urine. In many of the cases recorded the amount of sugar has been small: in a case reported by Burns not quite 1 per cent, and in another case between 1 and 2 per cent. Further, the symptoms continue if by strict diet the sugar can be made to disappear from the urine.

The above facts seem to indicate that the neuritis is not due directly to the presence of an excess of sugar in the blood. Auché has made experiments on the lower animals, and exposed the sciatic nerve to the action of different fluids containing sugar. His experiments show that sugar has only a slight action on the nerves, similar to that produced by water, and the changes in the nerves are probably due to some other cause—the poverty of the tissue in water, the general disturbance of nutrition, acetone, or some unknown chemical substance in the blood. Gowers believes that the neuritis is due to some toxic substance comparable to acetone, but not acetone, and thinks the fact, that the reduction of the amount of sugar formed has but little influence on the condition, suggests that the poison is not a product of the decomposition of sugar, but a material formed in place of sugar by some modification of the chemical processes that lead to the increased sugar production.

[1] BOUCHARD. "Sur la perte des reflexes tendineux dans le diabète sucré."—*Progrès méd.*, 1884, No. 41.

[2] MARIE ET GUINON. "Sur la perte du reflexe rotulien dans diabète sucré."—*Revue Médicale*, 1886, p. 640.

[3] AUERBACH. "Ueber das Verhältniss des Diabetes mellitus zu Affectionen des Nerven Systems."—*Deutsches Archiv f. klin. Med.*, 1887, Bd. XLI., p. 484.

[4] MASCHKA. "Ein Beitrag zur Symptomatologie des Diabetes mellitus."—*Wien. med. Presse*, 1885, No. 3.

[5] EICHHORST. "Beiträge zur Pathologie der Nerven und Muskeln. Neuritis diabetica und ihre Beziehungen zum fehlenden Patellar sehnenreflex."—*Virchow's Archiv.* Bd. CXXVII., p. 1.

[6] ROSENSTEIN. "Ueber das Verhalten des Kniephänomens beim Diabetes mellitus."—*Berlin. klin. Wochenschr.*, 1885, No. 8, p. 113.

[7] BUZZARD. *Lancet*, January 28, 1888.

[8] v. ZIEMSSEN. *Bayerische ärztliches Intelligenzblatt.*—(*Münchener med. Wochenschrift*) 1885, No. 44.

[9] v. HÖSSLIN. *Münchener med. Wochenschrift*, 1886.

[10] PRYCE. *Lancet*, July 2, 1887.

[11] LEYDEN. "Die Entzündung der peripheren nerven."—1888.

[12] ALTHAUS. *Lancet*, 1890. Vol. I., p. 455.

[13] CHARCOT. "Sur un cas de paraplégie diabétique."—*Arch. de neurologie*, 1890, Vol. XIX., p. 305.

[14] BUZZARD. *British Medical Journal*, June 21, 1890.

[15] AUCHÉ. "Des alterations des nerfs peripheriques chez les diabétiques."—*Archives de Méd. experimentale et d'anatomie pathologique*, No. 5, 1890.

[17] BURNS. "Ueber neuritische Lähmungen beim Diabetes mellitus."—*Berliner klin. Woch.*, No. 23, 1890.

[20] LECORCHÉ. "Troubles nerveux dans le diabète chez les femmes."—*Arch. de Neurologie*, Vol. X., p. 395, 1885; and Vol. XI., 1886.

[21] BERNARD and FÉRÉ. "Des troubles nerveux chez les diabétiques."—*Arch. de Neurologie*, Vol. IV.

[22] SALOMONSEN. Quoted in the *Annual of the Universal Medical Sciences*, Sajous, Philad., 1892.

[23] VERGELY. "Diabetic Zona."—*Le Progrès Médical*, September 26, 1891.

III.—THE DYSCRASIC FORM OF PERIPHERAL MULTIPLE NEURITIS: CHLOROSIS, MARASMUS, CANCEROUS AND OTHER FORMS OF CACHEXIA, VASCULAR DEGENERATION.

The slighter phenomena of peripheral neuritis are not infrequently met with in chlorosis, anæmia, especially the pernicious form, senile marasmus, and the cachexia of cancer. The patient suffers from numbness and tingling pains in the hands and feet, shooting pains in the lower extremities, and great muscular weakness. The electrical excitability of the muscles is often diminished, but the tendon reactions and the reflexes are generally maintained.

Cases of atrophic paralysis have been described in connection with *anæmia*, and their favourable issue suggested a peripheral rather than a central lesion. Thus an interesting case is reported by Lépine of a girl, æt. 20, who had suffered from severe anæmia for one year. On examination she was very weak, and presented the typical appearance and all the symptoms of marked anæmia. The hydræmic condition of the blood, and the great diminution in the amount of hæmoglobin, confirmed the diagnosis of *chlorosis*. About this time paralysis of the limbs was observed which had set in insidiously and at an unknown date. In the upper limbs the extensors of the forearms were chiefly affected, the supinators being relatively spared. The interossei and the thumb muscles were also implicated. In the lower limbs the anterior muscles of the legs were mainly paralysed, and the patient could not raise the toes or feet. The affected muscles were wasted, but the paralysis was greater than could be accounted for by the degree of atrophy. The electrical irritability of the muscles was diminished; the knee-jerks, diminished at first, were speedily abolished; the cutaneous sensibility remained normal. Eight months later there was great improvement in the power and nutrition of the muscles, but the knee-jerks were still absent; subsequently, however, a complete recovery ensued. In this case, as Lépine points out, the anæmia and nervous symptoms ran a parallel course, the latter subsiding as the condition of the blood improved.

The possibility of a cord lesion must not be overlooked in considering the paralysis found in association with anæmia. Lichtheim has reported three cases in which spinal symptoms developed during the course of pernicious anæmia of rapid progress. The symptoms were muscular weakness, ataxia, and sensory disturbances. The tendon reactions were maintained, but were less active than in health. The pupils reacted to light and accommodation. At the post mortem of two of the cases the posterior columns, especially Goll's tracts, were extensively diseased, but the peripheral nerves were healthy. The lateral and anterior columns in the cervical and dorsal regions presented small foci of disease, and slight lesions of the posterior roots in the lumbar region were also discovered.

Rosenbach examined the cerebrum, cerebellum, cord, and the spinal and sympathetic ganglia of starved dogs and rabbits, and found in all these parts of the nervous system an atrophic degeneration of the nerve cells, as well as changes in the neuroglia and vessels, but the nerve fibres were unaffected.

It is obvious that the paralyses which occasionally develop during the progress of chlorosis or pernicious anæmia require further investigation. The nutrition and functions of the spinal cord and peripheral nerves must suffer along with the heart and other organs, and hence it may be difficult to say how far certain nervous symptoms own a central or a peripheral origin. But apart from nutritive changes in the nervous system it must not be forgotten, as Dr. Gowers has suggested, "that imperfect tissue changes may generate a toxic agent capable of acting on the nerves, analogous to that assumed to be effective in diabetes."

Cancer.—The researches of Oppenheim, Siemerling, Klippel, and others have shown that peripheral neuritis is sometimes associated with cancer. Following up the investigations of these observers, Auché examined the nerves of ten cancerous subjects, and found in nine of the ten cases distinct evidence of parenchymatous neuritis. The more marked changes were found in the distal branches, while the nerve trunks and roots were healthy. In some of the cases no nervous symptoms were present during life, in other cases various sensations had been observed in the limbs, such as tingling and formication, sensations of cold and constrictive pains. The position of the cancerous mass appeared to bear no relation to that of the neuritis, and the influence of compression could be absolutely excluded, for the nerves of the upper limbs were found to be affected in cases where cancer was situated in the uterus or stomach. As to the causation of neuritis in connection with cancer, Auché believes that it is probably due to nutritive disturbances, and to the alterations of the tissues and their juices, which come on during the cachectic periods of cancerous tumours.

Senile and Atheromatous Neuritis.—It is probable that symptoms indicative of slight neuritis are not uncommon in old persons. Certainly degeneration of the peripheral nerves has been clearly demonstrated, post mortem, especially by the researches of Gombault. This degeneration may be partly explained by the malnutrition of old age, and partly by an imperfect supply of blood to the nerves owing to atheroma of the arteries. Gombault examined the external division of the dorsal digital branch to the great toe, and found in a large number of aged subjects, who had suffered from chronic disease, involving a protracted malnutrition of the tissues, that the myelin of the nerve fibres was much broken up and destroyed, but the axis-cylinder remained intact:—"segmental peri-axillary neuritis."

A moderate degree of claw hand, with atrophy of the interosseous muscles and lumbricales, and of the muscles of the thenar and hypothenar eminences, is not a very uncommon condition in cases of atheromatous degeneration and calcification of the radial arteries.

ROSENBACH. "Ueber die durch die Inanition bewirkten Texturveränderungen der Nerven-centren."—*Neurol. Centralblatt*, 1883.

LICHTHEIM. "Ueber Pathologie und Therapie der perniciösen Anämie."—*Neurol. Centralblatt*, 1887, p. 235, und 1889, p. 662.

LÉPINE (R.). "Anémie chronique grave; paralysie atrophique généralisée predominante dans les muscles extenseurs."—*Revue de Médecine*, 1887, 137.

OPPENHEIM und SIEMERLING.—*Archiv für Psych.*, 1887, p. 517.

GOMBAULT (A.). "Note sur l'état du nerf collatéral externe du gros orteil chez le vieillard."—*Neurol. Centralblatt*, 1890, p. 746.

AUCHÉ. "Des névrites périphériques chez les cancereux."—*Revue de Médecine*, 1890, p. 785.

JOFFY et ACHARD. "Atheromatous neuritis."—*Virch. Jahrb.*, Bd. XXIV.

IV.—SENSORY VASO-MOTOR AND TROPHIC NEURITIS. (1) THE NEURITIS FOUND IN CASES OF ATAXIA AND WHICH HAS BEEN NAMED NEURO-TABES PERIPHERICA.

Under this heading two groups of cases require discussion:—

A.—Cases of ataxia in which the main lesion is disease of the posterior columns of the spinal cord—that is, ordinary tabes dorsalis—but in which changes are also found in the peripheral nerves.

B.—Cases of ataxia in which the main or only lesion is disease of the peripheral nerves.

It is now well established, from the researches of Westphal, Pierret, Pitres and Vaillard, Oppenheim, and Siemerling, Déjérine, and others, that disease of the peripheral nerves, both cranial and spinal, is very frequently found in association with disease of the posterior columns in ordinary cases of locomotor ataxia. Déjérine has shown that in many cases there is a close correspondence between the distribution of anæsthesia and that of changes in the peripheral nerves; and it is also highly probable that the trophic lesions of the skin, bones, and joints, depend on the process of degeneration in the peripheral nerves. The question arises and has often been discussed, how far is the inco-ordination in any given case due to central or peripheral lesions.

The independence of these lesions is shown by the existence and combination of symmetrical cord lesions with unilateral neuritis and by the absence of any relation between the gravity of nerve lesions and the duration, extent, or gravity of cord lesions; thus, as Oppenheim demonstrated, considerable changes may be met with in the peripheral nerves at the onset of tabes when cord lesions are limited, and conversely the peripheral nerves may be intact when sclerosis of the posterior columns, of considerable extent, has existed for a long time. The latter point is substantiated by abundant evidence, and there cannot be a reasonable doubt that ataxia, in many cases, occurs as a direct consequence of disease of the posterior root zones.

We may now consider group B, and ask whether genuine ataxia ever occurs from lesions of the peripheral nerves alone, other parts of the nervous system being healthy. At first sight such a question appears to be superfluous, because numbers of cases of ataxia in connection with peripheral neuritis have been reported; but it is to be noted that in many of them the evidence of ataxia is not conclusive, and it is beyond question that the high stepping gait of multiple neuritis has often been ascribed to ataxia when it is really due to paralysis of certain muscles of the legs. It will be gathered from Dr. Ross's account of the motor disorders in alcoholic and diphtheritic paralysis that he did not believe in the existence of genuine ataxia as a consequence of peripheral neuritis. (See pp. 94, 252.)

A little consideration of the subject suggests either that many of the published observations are inaccurate, or that there is sometimes a real difficulty in discriminating between abnormal movements which are the result of inco-ordination, and those which depend on muscular weakness. To assign definite limits to the use of the term ataxia is not indeed such an easy matter as might at first appear. Accurate adjustment of muscular action is necessarily impaired by weakness or by spasm of any muscle employed in any particular movement, but such imperfections of movement ought not to be included under the term "ataxia." Ataxia or inco-ordination implies errors in the balance or equilibrium of the contraction of certain muscles required for a given movement, apart from alterations in their strength. And although paralysis or spasm may be found in association with ataxia, the latter, as in tabes dorsalis, frequently exists without the former. Nevertheless, there is sometimes a difficulty in deciding whether a defective movement is due to an alteration in the strength of certain muscles or merely to a want of proportion between their individual actions. In some cases of "writers' cramp," for example, where there is no obvious weakness or spasm of any of the muscles used in writing, it may be difficult to give a mechanical explanation for the muscular irregularities displayed in the attempt to write, and the same remark applies to motor defects that are sometimes met with in cases of multiple neuritis.

The difficulty referred to, however, is mainly limited to minor defects in muscular action, for as a rule a careful examination will enable us to decide whether certain defects in the movement of a part are due to inco-ordination or paralysis. Also when the two conditions are combined, as, *e.g.*, in ataxic paraplegia, the inco-ordination of movement is still recognisable unless paralysis is profound. Hence in the earlier stages of multiple neuritis, when the muscular weakness is moderate in degree, inco-ordination of movement should, if present, be readily detected.

Now Dr. Ross, if I understood him aright, stated that he had never seen ataxia in cases of multiple neuritis, and that observed defects in movement could all be explained by muscular weakness, perhaps occasionally combined with spasms. My own experience accorded with that of Dr. Ross, till quite recently, when the following case of alcoholic multiple neuritis came under my care, in which signs of ataxia were unmistakable.

The patient was a woman, aged 37, with a strong history of alcoholism. The mental condition was characteristic and the heart was dilated. Muscular hyperæsthesia was severe in both arms and legs. There was marked anæsthesia in the lower limbs, and partial anæsthesia in the hands and forearms. The sense of movement and position were also very defective; thus when the toes or feet were moved she felt nothing; she could tell, however, when the legs were raised from the bed, but was unable to say whether they were crossed or not. A weight of several pounds suspended from the foot or ankle was not felt, and when weights were placed on the feet patient was not aware of their presence. She could tell when a finger was moved but could not say which finger, she said she felt that something about her hand was moving but could not tell anything more, nor did she feel anything when her wrist or elbow was moved, but she could distinguish movements at the shoulder, and movements at the elbow were referred to the shoulder. The weight sense was lost in the hands as well as in the feet. The knee-jerks, the plantar reflexes, and in all probability the abdominal reflexes, were quite absent. As regards motor power, the patient was able to move any part of the lower limbs, but the movements were feeble, and the flexors of the ankles were especially weak; she could partially extend the wrists and fingers, but only by the exercise of great effort, and the power of grasp was almost completely lost. While there was considerable muscular weakness there was not complete paralysis of any movement. The patient was unable to stand or walk without assistance; when supported, her gait presented the most striking resemblance to that of typical tabes dorsalis; the legs were jerked forcibly forwards, and the feet brought down with a stamp; sometimes they became entangled, and were thrown hither and thither in the greatest disorder. It was particularly noticed that the anterior part of the advancing foot was raised; the gait, in fact, was totally unlike the ordinary high-stepping one of alcoholic paralysis.

The patient was treated at first with salicylate of soda and iodide of potassium, and subsequently with strychnine and massage; in two months time she was much better, and when I saw her the other day, just eight months after the appearance of the first symptoms of neuritis, the movements of the limbs were almost quite natural, but the knee-jerks were still absent.

Similar cases have been reported by Déjerine, Leyden, Dreschfeld, and others, in which multiple neuritis was found on post-mortem examination. Ataxic conditions have also been described in connection with diabetes in cases of lead and arsenic poisoning, and as following diphtheria, measles, and other acute diseases.

In most of the reported cases of ataxia occurring in connection with peripheral neuritis, other motor and sensory symptoms were present, and this is to be expected from the constitution of the mixed peripheral nerves. Does ataxia ever occur as the only symptom in cases of multiple neuritis? We know that this may happen when disease is limited to certain tracts in the spinal cord. Are there fibres in the peripheral nerves which, when picked out by disease, give rise to inco-ordination of movement, and to that alone? It is impossible to give a complete answer to such a question at present, especially from the pathological side. Certain cases have been reported in which muscular weakness and sensory disorders have been quite insignificant, while ataxia has been pronounced, and where complete recovery has strongly suggested peripheral rather than cerebral lesions. These conditions obtained in the following case of acute ataxia recorded by Leyden.

A man, æt. 55, after working in a cold room when his feet were sweating, experienced creeping sensations in the legs and numbness in the feet. Five days later, after a hard day's work, these sensations returned, and for the first time his legs felt weak, and on the next day he could only walk with the help of a stick. He was compelled to go to bed. When lying down he was free from pain, and the abnormal sensations in the legs became less troublesome. His condition on the eighth day from the onset of symptoms, when he came under Professor Leyden's care, was as follows:—

All the symptoms were limited to the lower extremities, an examination of the cranial nerves, the upper extremities, and the thoracic and abdominal organs revealing nothing abnormal. The motor power of the lower limbs was not impaired, the patient could perform all the movements at the different joints perfectly well, and he offered strong resistance to passive movements. All the movements of the lower limbs presented marked ataxia; with closed eyes he was unable to touch the knee of one limb with the heel of the other. He was unable to stand, and when supported his gait was distinctly ataxic; Romberg's phenomenon was also present. Tactile sensibility was only slightly impaired, but painful sensations were severely felt; and he complained of numbness and creeping sensations in the extremities. The knee-jerks were abolished; the cremasteric reflex could not be obtained on the left side, and was diminished on the right side. He was unable to pass urine; when withdrawn by a catheter it was found to be alkaline, but did not contain albumen or sugar.

A week later urine was passed spontaneously, but only in small quantities at a time: the patient could walk better, but ataxia was still conspicuous, and he complained of darting pains in the feet. Subsequently, improvement went on rapidly, and in a month from the onset of the first symptom the knee-jerks had returned, the functions of the bladder were normally performed, and with the exception of slight unsteadiness in walking the patient was almost quite well. The most important antecedent was syphilis, from which the patient had suffered in early life, but Leyden is inclined to attribute the symptoms to a peripheral neuritis, which had been started by exposure to damp and cold.

Obviously there is a difficulty in locating the lesion in some cases of ataxia. Rapid loss of the knee-jerks and of the muscular sense, in association with sensory disturbance and motor weakness, speak for a peripheral rather than a central lesion; but duration appears to be the only certain test, and speedy recovery may be regarded as strongly in favour of peripheral neuritis. It is highly probable, then, from the clinical and pathological evidence adduced, that cases of ataxia presenting no striking difference from ordinary tabes dorsalis may result from disease of the distal parts of the spinal nerves, while the following abstract of the well-known case reported by Hughes Bennett suggests that disease limited to the proximal ends of sensory nerves may also produce ataxia:—

A gamekeeper, 48 years old, having previously been in good health, began to suffer about nine months before he came under observation from attacks of shooting pains in the legs, inability to walk properly, and a sensation of numbness in the feet. All these symptoms slowly increased in severity. On examination the general health and all the functions and organs of the body were found to be normal with the following exceptions:—There was slight nystagmus, also occasional giddiness, but otherwise the special senses and the cerebral functions were normal. The grasp of the hands was weak, and the delicate actions of the fingers were slightly inco-ordinated. The lower limbs presented all the typical characters of advanced ataxia, the inco-ordination being markedly increased in the dark or with the eyes closed. The knee-jerks were totally absent, and the plantar reflexes were diminished. In the legs and feet the cutaneous sensibility to touch and pain was diminished and retarded. The patient complained of a constant dull aching in his back and lower limbs, and the latter were the seat of frequent attacks of lancinating pains. The muscles were nowhere atrophied, and their mechanical irritability and electrical reactions were unimpaired. During a residence of two and a half months in the hospital all these symptoms somewhat rapidly increased, and the patient died of exhaustion. On post-mortem examination the essential lesion was found to consist of a series of isolated sarcomatous tumours situated chiefly in the posterior portion of the pia mater of the cord. These involved the posterior spinal roots, but without causing their degeneration; indeed the only histological change that could be detected was "unusual swelling of the white substance of Schwann." The state of the nerve trunks with their peripheral terminations in the muscles and skin were not examined. The tumours were most numerous in the lower dorsal and lumbar regions. The posterior columns of the cord were everywhere perfectly normal. A sarcomatous growth was also found in the floor of the fourth ventricle. It extended downwards, and surrounded the central canal.

It is unfortunate that the cutaneous and muscular nerve endings escaped examination, but it is clear, as Dr. Bennett points out, that the primary disease did not originate there, and it seems fair to regard the case as an important pathological link between the classical lesion in the posterior root zones and the cases of "nervo-tabes périphérique," described by Déjérine.

A study of the relation of ataxia to peripheral neuritis brings out two points:—

1. That ataxia is a rare symptom in cases of multiple peripheral neuritis, even when cutaneous and muscular sensibility are profoundly affected.

2. That it occasionally occurs when signs of muscular weakness and of diminished cutaneous sensibility are slight or absent.

It is probable that the presence of ataxia in cases of multiple neuritis points to disease of sensory muscle-nerves, but it is very remarkable that extreme degeneration of terminal nerve-fibres to both skin and muscle has been found in cases which presented no signs of muscular inco-ordination during life. It is highly important in all cases of multiple neuritis which come to post-mortem examination, that the results of histological investigation as to the condition of nerves to muscle and skin should be carefully studied in relation to any indications of ataxia exhibited during life.

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(2) THE VASO-MOTOR NEUROSIS, FIRST DESCRIBED BY WEIR MITCHELL UNDER THE NAME OF ERYTHROMELALGIA.

This disease is much commoner in the male than in the female. It usually comes on after prolonged exertion, such as tramping, or some constitutional disease, or gonorrhœa. A slight degree of it is occasionally met with in association with subacute rheumatism (see p. 345), and is especially common in cases of gonorrhœal rheumatism. The prominent symptoms consist of redness and pain of the affected limbs. "The pain usually begins in the ball of the great toe, or in the heel, and often extends over a great part of the sole, and may reach the dorsum of the foot and leg. It is generally limited to circumscribed areas of one or both soles, and does not extend beyond. It is felt at first towards night, and is relieved by the night's rest; it is increased by walking, the erect posture, or even by allowing the foot to hang down. The pain is felt at first as a deep aching, but after a time it becomes of a burning character, and is then aggravated by warmth, and relieved by cold and the recumbent position. The most characteristic symptoms of the affection, however, is a flushing of the painful area, which comes on with exertion, or when the feet are allowed to hang down. 'The foot,' says Dr. Mitchell, 'gets redder and redder; the veins stand out in a few minutes as if a ligature had been tied around the limb, and the arteries throb violently for a time, until at length the extremity turns of a dark purplish tint. In the worst cases, when the patient is at rest, the limbs are cold, and even pale.' In aggravated cases the pain is so severe as to render walking all but impossible, and when persisted in intense redness and swelling are occasioned; the patient sleeps with uncovered feet, and in the worst cases crawls on his hands and knees, or is carried about in order to avoid placing his feet on the ground." The disease is at times progressive, and in the later stages it extends to the hands. In a case under the care of Dr. Ross two or three years ago, the hands were first, and chiefly, affected. As Dr. Ross has pointed out, and as I have frequently observed in gonorrhœal cases, a tender spot is present over the centre of the heel, and painful points may be found by pressing between the heads of the metatarsal bones over the bifurcation of the branches of the plantar nerves for the toes.

Evidences of paralysis with wasting are sometimes to be found in the small muscles of the feet and in those of the legs, the faradic contractility of which may be diminished. In the case in which the disease began in the hands, the muscles of the thenar and hypothenar eminences as well as the interossei and lumbricales were distinctly atrophied, and gave a diminished reaction to the faradic current.

At a meeting of the Philadelphia Neurological Society, Dr. C.

Woodnut mentioned the case of a man, aged 50, who, previously healthy and free from suspicion of alcohol or syphilis, began to suffer with peculiar pains in the second toe and along the inner side of the left foot, and after a year in the third and fourth toes, and subsequently in the big toe. In the course of another year the pains had spread to the second, third, and fourth toes of the right foot. The pain was at first burning, then violent and tearing in character. It was associated after a time with a reddish violet discolouration of the skin, which extended as far as the ankle and disappeared on pressure. Similar spots of discolouration subsequently appeared on the left leg and in the middle of the back. Exercise and warmth aggravated the pains. There were also occasional lightning pains in the lower limbs, along the spine, and radiating from it towards the brachial plexus. The cutaneous sensibility of the left leg was diminished, except over the discoloured patches where there was hyperæsthesia. Treatment with rest, massage, electricity, and the administration of various drugs affording no permanent relief, the left second toe was amputated, and this operation was followed by a diminution of the tenderness of the whole of the left foot.

In Dr. Woodnut's case other parts than the feet were the seat of pain, congestion and tenderness; and in a patient under the care of Auché and Lespinasse the face was affected as well as both hands and both feet. The patient, a man, aged 30, was a bleacher in a paper works; he worked the greater part of the day in a hot atmosphere, and then cooled himself by washing in cold water. The attacks of pain and congestion were accompanied by a local rise of temperature, and by increased frequency of the pulse and respirations. Sensibility, motility, and electrical reactions were normal.

The testicle also became subject to attacks of swelling and redness, the condition being associated with very painful erections.

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(3) Raynaud's Disease.

In discussing the relationship of Raynaud's disease to peripheral neuritis, it is unnecessary to give a complete description of the disease or to discuss its affinities with hæmoglobinuria and sclerodermia, but it is essential to keep in view the characteristic features that underlie the somewhat varied group of symptoms comprised under the term.

The condition is essentially a paroxysmal one, characterised by the presence of skin lesions which are either temporary or permanent. Three stages or forms of the disease may be distinguished. (1) The stage of local syncope, in which there is a paroxysmal condition of pallor of the extremities. (2) The stage of local asphyxia, in which there is a paroxysmal condition of blueness of the extremities. (3) The stage of local gangrene.

The pallor, the lividity, and the gangrene are usually symmetrical in distribution, but by no means invariably so. They may affect the fingers, the toes, the tips of the ears, or nose. In the simplest cases there is a series of attacks of local syncope or local cyanosis in which the affected parts become cold and present either a dead white or a livid appearance; their sensibility is blunted and their motor power diminished, and they are frequently the seat of severe pains resembling those of frost bite.

These colour changes are excited by very slight variations in the surrounding temperature, and tend to become "marked and prolonged out of all proportion to the stimulus" which starts them.

In severe cases the condition of local syncope, or more commonly that of local cyanosis, is succeeded by spontaneous gangrene. This is generally of the dry variety, and in a large number of cases is remarkable for its limited and superficial distribution.

Raynaud explains these facts on the hypothesis that there is a spasmodic contraction of the capillary vessels. He points out that in the simplest cases of vascular spasm the exaggerated contraction of the capillaries drives the blood before it; the extremities become pale, withered-looking, and insensible. This is the "dead finger." But this phenomenon does not persist long enough for gangrene to follow; to contraction succeeds relaxation, the circulation is re-established, and everything returns to the normal state, after a period of reaction more or less painful. "Such is *local syncope*, in which the venules participate in the contraction of the arterioles. Local asphyxia is a more advanced condition. After an initial period of spasm there occurs a period of reaction, but it is incomplete reaction. The vessels which return first to their primary calibre, or even beyond, are those which present in their structure fewest contractile elements, viz., the venules. When these are opened, the arterioles being still closed, the venous blood, which had at first been driven back into the great venous trunks, flows again into the finest vascular divisions, and then the extremities will take on that tint, varying from blue to black, which is a certain index of the presence of venous blood in the capillary network.*

"The condition is susceptible of many degrees. At one time everything is comprised in local asphyxia pure and simple; at another, each attack having a longer duration, the tendency to gangrene is more pronounced; bullæ form with very small sloughs; then at the moment when gangrene is on the point of becoming confirmed the parts revive momentarily, to be soon afterwards attacked in the same way; and this may go on for years. Finally it may happen, although much more rarely, that the capillary spasm comes on all at once, with an intensity and duration altogether extraordinary. Syncope and local asphyxia succeed one another rapidly; the venous blood becomes insufficient to nourish the parts, the colour becomes deeper, small blood-stained infiltrations take place through the walls of the venules; these walls themselves may become granular—in one word, there is confirmed gangrene, and gangrene which may go on to the fall of many ends of fingers or toes.

"But on final analysis all these varieties of functional lesions, which may each present an infinity of shades, realise always one or other of the two conditions essential to dry gangrene—that is to say, absence of blood or presence of blood unsuitable to nutrition."

The hypothesis of local vascular spasm receives strong support from a case reported by Raynaud in his later researches. It was that of a man aged fifty-nine, who in the intervals between ordinary attacks of local asphyxia suffered from dimness of vision, especially in the left eye; and on ophthalmoscopic examination it was found that there was marked narrowing of the lumen of the central artery of the retina and of its branches, and that the veins presented very striking dilatation and pulsation. The same characters were present to a less degree in the other eye. During the period of cyanosis of limbs the pulsations in the veins continued, and the arteries did not recover their normal calibre in all their extent. Partial diminutions of calibre were seen at times, so as to render portions of them filiform. Contractions were seen to form, persist for a time, then disappear, and contractions were then seen in other arteries. The correctness of these observations has since been confirmed, and Dr. Barlow has shown that definite contractions of both arterioles and venules may occasionally be seen in the extremities. In a well marked case of Raynaud's disease occurring in a little girl he observed a remarkable phenomenon in some of the veins on the back of the affected hand. During one attack "they became quite monilifirm—that is to say, there was an alternation of small dark swellings with narrow almost colourless intervals between them, and under observation the dark swellings gradually altered their position along the course of the veins, pointing to a varying contraction of the walls of these vessels."

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* This and the other quotations are taken from Dr. Barlow's translation of Raynaud's memoir.

Raynaud sums up his view of the pathology of the disease by saying: "That in the present state of our knowledge local asphyxia of the extremities ought to be considered as a neurosis characterised by enormous exaggeration of the excito-motor energy of the grey parts of the spinal cord which control the vaso-motor innervation."

It is obvious that this view comprises two assumptions, viz.: (1) There is disturbed vaso-motor innervation; (2) That this depends on altered energy of the grey matter of the cord. The first assumption is strongly supported by all the evidence in our possession, and it is pretty generally admitted that whatever may be the exact pathology of the local gangrene, the phenomena of local syncope and local cyanosis are the direct results of spasm of the capillary vessels. But the second assumption is merely a hypothesis, and calls for discussion.

Arterial spasm may be produced by stimulation of (*a*) certain parts of the cerebral grey matter; (*b*) the vaso-motor centre in the medulla; (*c*) the subordinate centres in the cord; or (*d*) the vaso-motor nerve fibres which are said to run in the mixed peripheral nerves..

A discussion, then, of the pathology of Raynaud's disease resolves itself into a discussion as to which part of the vaso-motor nerve tract is primarily affected, and as to the nature of the stimulant and the method in which it is brought to bear on this department of the nervous system.

Information on these points is to be looked for—(1) From the results of post-mortem examinations; (2) from a consideration of clinical observations on the disease in question, and on other affections presenting similar symptoms.

Pathological Evidence.—The following are the chief cases in which the nervous system was examined. Mounstein's case.—A man, aged fifty-one, had gangrene of the right foot. "The gangrene had led to the separation of the first, second, and fifth toes, whilst the third and fourth were isolated, but the skin over all the toes was involved in the gangrenous process as well as that covering the heel, the inner side of the foot, and the dorsum." Amputation of the right leg in the upper third was performed, but the patient succumbed a week later with a high temperature. "The vessels generally of the lower extremity were free from abnormal contents; only in the capillary vessels adjacent to the gangrenous focus were microscopic hyaline thrombi present. The posterior tibial artery showed many calcareous plates, but no thrombi adherent to them. The posterior tibial nerve was greatly thickened in its lower part; microscopic investigation showed great wasting of the myelin, with collapse of Schwann's sheaths, and chronic inflammatory proliferation of the interstitial connective tissue, especially in the parts close to the gangrenous area. The nerves in the left sound lower limb showed similar changes to those in the gangrenous limb. The nerve roots of the lumbar region were only affected with neuritis on the right side. Brain and cord were markedly anæmic, and the examination of the viscera gave negative results."

Dr. Barlow remarks that "the clinical history of this case is too meagre to allow of its being placed in Raynaud's group, but the double-sided affection of the nerves more extensive on the gangrenous side is very suggestive."

Cases reported by Pitres and Vaillard: (1) A woman, aged 24, of feeble intelligence from childhood, began to suffer at the age of 18, from tremors and stiffness of limbs until at length walking became impossible; the lower limbs passed into a state of extreme contraction, and the patient was bedridden and demented.

After a time the feet were noticed to be cold, blue and insensitive; they gradually became gangrenous; the left foot underwent spontaneous amputation, and the right was all but separated. Numerous eschars appeared on various parts of the body; many of them suppurated, and the patient died from exhaustion. The arteries of the lower limbs were free from adherent thrombi, and the veins were quite healthy. The skull was greatly thickened and there was hydrocephalus. The dorso-lumbar part of the cord presented a slight diffuse sclerosis, which affected the whole of the antero-lateral columns and the whole of the posterior columns except their anterior fifth. The spinal ganglia and nerve roots were natural. The nerves of the upper limbs and those of the thigh were also normal; but the anterior and posterior tibial of both sides presented changes characteristic of degenerative neuritis; the changes were of varying extent but were fairly symmetrical.

(2) A woman, aged 56, who had been subject to great hardships, suffered for six months from a sensation of considerable fatigue, and found that she no longer felt the soil on which she trod. Then bullæ formed on the soles of her feet, and she began to suffer from obstinate diarrhœa. The feet became swollen, painful, and covered with reddish

patches on the dorsal surface. Fresh bullæ formed, the feet were perfectly cold, and anæsthesia on the left side extended up to the ankle, on the right side to the middle of the tarsus. The line of separation formed at this level on both sides, but the patient died from exhaustion and diarrhœa before actual separation had taken place. Post-mortem showed neuritis of the plantar and tibial nerves, but the vessels of the limbs were natural, and the brain, spinal cord, and viscera were also natural.

The authors point out that in cases of gangrene of embolic origin no peripheral neuritis is found, and they regard the peripheral neuritis in their cases as the cause of the gangrene, and hold that most cases of Raynaud's gangrene are of like origin.

Wiglesworth has described a case of very extensive peripheral neuritis in a woman aged 26, who was the subject of epileptic dementia and of chronic Bright's disease, and in whom repeated attacks of spontaneous gangrene of fingers and toes had occurred. There was also extensive atrophy of the muscles of both hands.

The nerves examined were the following:—sciatic, internal popliteal, external popliteal, posterior tibial, median and ulnar on each side, and the right musculo-spiral—thirteen in all. The great majority of these nerves presented well-marked changes, and none of them were wholly unaffected. Briefly, the changes consisted in overgrowth of the fibrous elements of the nerve, with atrophy and degeneration of the nervous elements. The changes were more advanced at the distal than at the central ends of the nerves.

The spinal cord presented but slight changes; there was, perhaps, a little general thickening of the neuroglia, and the cells forming the posterior vesicular columns of Clarke were somewhat altered in shape.

Dr. Wiglesworth believes that the localised symmetrical gangrene in his case was directly dependent upon the changes in progress in the nerves.

In contrast to this case may be mentioned one under the care of Dr. Barlow, in which the peripheral nerves of a limb affected with massive gangrene were carefully examined, and found to be quite normal. The patient was a man aged 46, who had suffered for seven years from symmetrical paroxysmal vascular disturbances in the lower extremities. The attacks were at first confined to the cold weather, but gradually became chronic, although liable to exacerbations when the patient was exposed to cold. In the early attacks the feet were painful and cold; in the later ones they became blue, and the left foot, which had always suffered most severely, ultimately passed into the condition of moist progressive gangrene. The deep tissue of the foot became involved, and suppurating bullæ appeared on the leg and thigh. The condition becoming desperate, Mr. Beck amputated the limb in the middle third of the thigh. The anterior and posterior tibial and the plantar nerves were examined, as well as portions of muscle, with some of the smaller nerve twigs. There was no obvious increase of connective tissue in perineurium or endoneurium, the axis cylinders were well marked, and there was no segmentation of myelin. "We may say pretty confidently that in this case there is no evidence of peripheral neuritis." The arteries were thrombosed; there was some recent thickening of the outer and middle coat, and "a remarkable contortion and infolding in some sections of the elastic lamina," which seemed to have followed upon the shrinking of the thrombus. There was no atheroma and not a trace of calcification. The veins also showed some thickening of their walls. Dr. Barlow points out that "the early history of this case is not conformable to the supposition of a permanent block in any of the principal arteries, and that the very marked improvement which followed the peripheral stimuli of galvanism and shampooing could hardly have been expected if gross changes in the walls of the arteries had been present."

A girl, aged 17, under the care of Dr. West, suffered for about one year from typical attacks of local syncope and local cyanosis of the hands and feet. She was admitted to hospital, and, in addition to a peculiar eruption on the face, the toes and fingers presented a purplish, harsh, swollen, desquamating appearance. There were also superficial blebs on the thumb, and on some of the fingers, from which a milky fluid escaped. The girl died from an acute attack of pneumonia. The radial artery at the wrist, the median nerve at the wrist as well as the radial, were examined microscopically and found normal, no abnormal change was found in the medulla, and with the exception of pneumonia of the right lung the result of post-mortem examination was entirely negative.

Dr. Affleck has reported in detail two interesting cases of Raynaud's disease. In one of the patients typical attacks of pain and local cyanosis were followed by gangrene of the feet, the gangrene being so severe in the left foot that amputation was necessary. The blood vessels and the internal plantar nerve were submitted to careful examination. The former were quite healthy, but sections of the latter furthest removed from the diseased parts showed extensive neuritis and degenerative changes.

An important case of symmetrical gangrene has been recorded by Dr. Rakhmaninoff. The patient, a young man aged 17, was admitted to hospital suffering from gangrene of both feet and the lower parts of the legs. There was no history of syphilis, and no hereditary tendency to nervous diseases. His present illness dated from an attack of typhus fever two years previously. He became anæmic, suffered from a feeling of coldness and numbness in the hands and feet. The numbness was followed by twitching in the muscles of the hand and forearm, together with lancinating pains. Subsequently he had pains in the thorax and abdomen, and became liable to exaggerated sweating of the whole body. Three days before he came into hospital he had a sensation of cold in the upper extremities, accompanied by piercing pains; the cold feelings were replaced at night by an acute and insupportable sensation of heat. At the same time the patient noticed that the backs of his feet were red and swollen; the redness and swelling spread upwards, while the lower parts of the feet became of a bluish livid colour. A few days later a line of demarcation formed around the ankles, and it was deemed advisable to amputate the legs a little below the knees. The wounds healed quickly, but the patient became worse, and suffered from cough and pain in the left side; he soon became comatose, and died about sixteen days after the operation.

At the autopsy, pleurisy and pneumonia were found, and the spleen was large and diffluent.

Rakhmaninoff examined the chief nerves in the upper and lower limbs, as well as the small branches to the skin and muscles, and found in all of them evidence of degenerative neuritis. The changes were more pronounced in the nerves of the lower than in those of the upper extremities, and in the small branches than in the main trunk. The myelin was broken up into globules and small granules; in many places Schwann's sheath was quite empty. Segmentation and disappearance of the axis cylinders were also to be seen. The tissue around the nerve fasciculi was hypertrophied, and was rich in fat cells and collections of corpuscles containing brownish pigmentary granules; it also contained vessels with thickened walls and narrowed or obliterated lumens. The muscles of the ball of the thumb showed a slight narrowing of their muscular fibres and a proliferation of nuclei, their striation, however, being preserved. The spinal cord was quite normal.

Thus in this case there was a disseminated multiple neuritis, the nerves of the upper as well as those of the lower limbs being affected. This neuritis appeared to be of infectious origin, having started immediately after an attack of typhus fever. Alcohol and other well-known causes of neuritis could be absolutely excluded. Rakhmaninoff believes, too, that the neuritis was the cause of the symmetrical gangrene. He draws particular attention to the thickened vessels, but is unable to decide whether they ought to be regarded as secondary to the neuritis or as primary and the direct cause of it.

If now we leave out of consideration the case reported by Mounstein as being a doubtful example of Raynaud's disease, there remain seven cases in which the pathological changes may be analysed. Of these the peripheral nerves were normal in two, but showed marked changes in five. The spinal cord was examined in five cases; it presented slight changes in one case, a diffuse sclerosis in another case, and was healthy in the remaining three cases. The arteries were found to be normal in four, and altered in two cases.

It appears, then, that as regards gangrene in Raynaud's disease, lesions of the peripheral nerves are much commoner than lesions of the brain, cord, or vessels. But that they are not essential to the disease is clearly shown by the cases reported by Barlow and West, in which the peripheral nerves were quite healthy. Barlow's case is particularly important on account of its detailed clinical history, which proves that the case was a typical instance of Raynaud's disease, and of the careful examination of the nerves and vessels. The trunks, as well as the smaller branches of the nerves going to the gangrenous foot, were found to be perfectly normal; there was, in fact, no evidence whatever of peripheral neuritis. The case is, then, sufficient in itself to upset the doctrine that Raynaud's gangrene always depends on a peripheral neuritis. The arteries in Barlow's case presented changes, but in other cases of Raynaud's gangrene they have been found unchanged, so that arterial lesions can also be excluded as essential causes of the disease. It should, however, be noted that when the arteries were normal the peripheral nerves were diseased, and *vice versâ*; the only exception is West's case, but here the condition of the nerves was not fully investigated.*

Clinical Evidence.—(1) Vaso-motor disturbance occurs in consequence of lesions of single nerves, and is met with in conditions such as alcoholic paralysis known to depend on multiple neuritis. In the former case vascular disturbance and skin lesions affect the territory supplied by the particular nerve, but these changes do not occur paroxysmally, as in Raynaud's disease, and need not be further considered. In alcoholic paralysis evidence of vaso-motor spasm is common enough (see p. 157). At an early period of the disease the fingers and toes become cold, dead, and white, or, at times, quite livid, while in severe cases gangrene may develop. It is highly probable that these phenomena, which are identical with those that constitute Raynaud's disease, are the result of multiple neuritis—but there is no direct proof of this, and it is possible that while the paralytic phenomena of alcoholism are due to disease of the peripheral nerves, the vaso-motor disturbances are related to co-existing changes in the grey matter of the cord.

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* In a valuable case of Raynaud's disease, reported by my friend Mr. J. Collier, the vessels and nerves going to the gangrenous part were found to be normal, but the condition of the small branches of the nerves is not mentioned; also it is stated that the small vessels nearest the slough showed slight endarteritis.

(2) The ordinary symptoms of peripheral neuritis, viz. :—neuralgic pains, anæsthesia, muscular weakness and wasting, are often present in conjunction with vascular disturbances in cases of Raynaud's disease. Sometimes the initial pains are very severe and the subsequent anæsthesia may be well marked, both as regards distribution and degree (see cases reported by Coupland, Stevenson, and others). But none of the symptoms enumerated are constant or essential to Raynaud's disease; when they are present their associations and distribution often testify to the existence of peripheral neuritis, but it must be admitted that they—that is, the ordinary symptoms of peripheral neuritis—are as often absent as present.

Summary.—The cardinal feature of Raynaud's disease is the occurrence of vascular disturbance in peripheral parts on exposure to cold. Paroxysmal attacks of local syncope or cyanosis, succeeded or not by local gangrene, are often met with quite apart from the ordinary symptoms of peripheral neuritis. Sometimes, however, the latter are unmistakably present, and sometimes marked changes in the peripheral nerves going to the affected parts are found on post-mortem examination. But the inconstancy both of the clinical and pathological evidence of peripheral neuritis points to some other cause for the explanation of the typical phenomena of Raynaud's disease. The selective action of different poisons is well known; thus lead has a striking affinity for motor nerve fibres, alcohol for both sensory and motor, and possibly for vaso-motor fibres, and there may be a form of multiple neuritis in which the vaso-motor nerve fibres are alone or mainly picked out. Should the last variety be established by future investigation, then it might possibly be correct to classify erythromelalgia and Raynaud's disease as examples of the rare vaso-motor form of peripheral neuritis, but at present it appears to me impossible to suggest a better hypothesis than that brought forward by Raynaud. His view may be summarised and expanded as follows :—

The early paroxysmal phases of Raynaud's disease are instances of reflex action produced with abnormal ease. Cold, the chief cause, irritates the morbidly active vaso-motor centres in the brain and cord. The result is spasm of the capillary vessels. Repeated and protracted attacks of vascular spasm may initiate actual disease of the walls of the small vessels. The peripheral neuritis which is sometimes present in Raynaud's disease must be regarded as an epiphenomenon; it may be the direct result of arterial disease as suggested by Rakhmaninoff's case, or occur independently of this, the nerve degeneration being due either to imperfect supply of blood, owing to vascular spasm, or to changes in those central cells which preside over the nutrition of the peripheral nerve endings.

To put the matter in another way, we may say that the clinical phenomena of erythromelalgia and Raynaud's disease, in their association and distribution, are somewhat analogous to those produced by peripheral neuritis, but that they depend on such a condition, even giving the widest possible meaning to the term, is far from being demonstrated.

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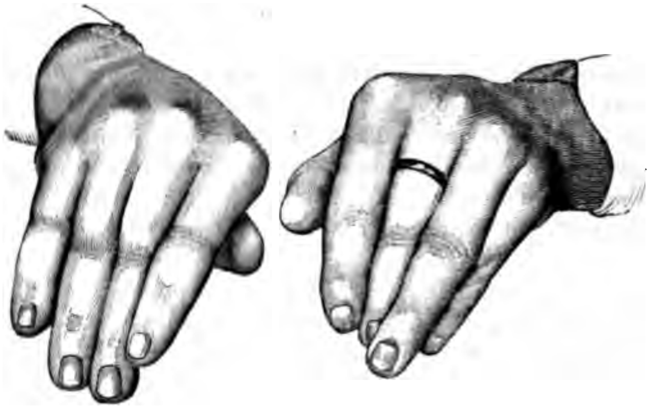
V.—THE IRRITATIVE FORM OF NEURITIS, IN WHICH SPASM PREDOMINATES OVER PARALYSIS.

Muscular spasm may be taken as an indication that some portion of the motor tract is in an abnormal condition. In the consideration of all motor phenomena it is convenient to regard the motor tract as composed of two parts, an upper and a lower segment. The upper segment comprises the motor cells in the cerebral cortex and the pyramidal fibres which extend between these cortical centres and the anterior horns of the spinal cord or the corresponding nuclei in the medulla and pons. The lower segment includes the bulbar nuclei and the anterior horns, together with the motor fibres which extend from them to the muscles.

A lesion situated in any portion of the upper segment of the motor tract gives rise to a spastic paralysis—that is, to a paralysis associated with rigidity or spasm of muscular tissue. A lesion situated in any part of the lower segment gives rise to an atrophic paralysis, in which weakness is combined with relaxation and wasting of muscular tissue; and how far active spasm may result from irritation of this tract cannot be precisely stated. As examples of spastic paralysis may be mentioned an ordinary hemiplegia from a lesion of the pyramidal fibres in the internal capsule and a spastic paraplegia as a result of disease of both pyramidal tracts in the spinal cord. Now it is to be noted in cases of spastic paralysis: (1) That the condition of the affected part is much the same whether the lesion be situated in the cortical motor areas, in the pyramidal fibres, or in their terminal ramifications in the grey matter of the cord. (2) That the relation between spasm and paralysis of muscle is very variable. They may be associated together in almost equal proportions, but frequently it will be found that while a limb exhibits a fair amount of power its muscles feel as hard as boards, and each movement is interrupted by violent spasm; in other cases slight spasm, indicated by a very slight increase of resistance to passive movements, is associated with considerable weakness. (3) That the condition of the muscles as regards rigidity is largely influenced from time to time by irritation of grey matter in consequence of external stimuli to sensory nerves; and I believe, from the evidence of two remarkable cases, that very rarely intense spasm of the legs may be started by peripheral irritation, and may become a permanent condition apart from any evidence of paralysis or other indication of organic disease of the nervous system.

Increased muscular tonus then is one of the distinguishing features of affections of the upper segment; diminished muscular tonus of affections of the lower segment of the motor tract. We may now ask does muscular spasm ever occur in cases where a lesion is limited to some portion of the lower segment? First with regard to the *spinal nuclei*, will irritation of the motor cells in the anterior horns, and restricted to them, produce muscular spasm? Physiologists do not appear to be agreed as to the excitability of any part of the cord to direct electrical and mechanical stimuli; it is stated that the motor centres can be excited by blood heated above 40°C., or by asphyxiated blood, or by certain poisons, as strychine (Landois and Stirling). The knowledge derived from a study of cord diseases does not lend any support to the view that spasm may be caused by a lesion limited to the anterior horns. How rare, for example, is it to meet with any signs of local muscular rigidity in infantile paralysis, even at the onset of the affection? And when it is present in this disease it is probably due to an associated lesion, as meningitis. Twice in infantile paralysis I have seen slight rigidity and exaggeration of the knee-jerk of the non-paralysed leg, but these symptoms in all probability pointed to implication of the terminal fibres of the pyramidal tract.

Secondly, as regards the *peripheral nerves*. Irritation of motor nerves by the electric current or other stimuli tends to produce muscular spasm. Has disease a similar effect? The answer is that spasm of the ocular muscles may result from basal meningitis in consequence of irritation of the motor nerve trunks; that facial spasm may be caused by a tumour pressing on the facial nerve; and that muscular cramps are common in the early stages of conditions, such as alcoholic paralysis, that are known to depend on peripheral neuritis. Further, in the later stages of multiple neuritis, when paralysis is pronounced, irritative motor phenomena are by no means always absent. In the well marked case of alcoholic paralysis already referred to on p. 380, the position of the hands was much like that seen in tetany. The dropped wrist was the result of paralysis of the extensor muscles, but the flexion of the first phalanges of the fingers and the extension of the terminal ones were mainly caused by spasm of the interossei; this was proved by the firmness with which the fingers were pressed together and by the resistance experienced on trying to separate them or to extend them at the metacarpo-phalangeal joints. The fingers, too, were never completely at rest, and their quivering movements appeared to be due to intermittent contractions of the interossei.



Let us now consider whether it is possible to admit the existence of an irritative form of peripheral neuritis, which is clinically expressed by muscular spasm, and by no other prominent symptom. We know that paralysis of the extremities may result from disease of the peripheral nerves. Are there cases in which a spasmodic condition of the extremities can be attributed to morbid irritation of a number of motor nerve fibres? This question naturally suggests tetany, a disease, the clinical features of which present certain affinities with those of multiple peripheral neuritis.

Tetany may be described as a condition characterised by a peculiar tonic spasm of the extremities, which is generally symmetrical and lasts for a variable time. The wrists are slightly flexed while the attitude of the hand resembles that of the accoucheur, the fingers being approximated, flexed at the metacarpo-phalangeal and extended at the other joints; the thumb is extended and strongly adducted, its tip being applied to the radial side of the forefinger, and the palm is made still more hollow by an approximation of its inner and outer borders. The characteristic change in the foot is extreme flexion of the toes, which sometimes overlap one another; the forepart of the sole is often contracted so as to become remarkably concave, with a median furrow, due to approximation of the outer and inner margins. The hands and feet are usually painful, tender and swollen, and the wrist and dorsum of the foot often red and shining. The rigidity may spread to other parts of the limbs, and very rarely to all the muscles of the body. These spasmodic attacks are usually preceded and often followed by tingling or burning sensations in the extremities. The sense of touch may be impaired, but there is rarely much diminution of cutaneous sensibility; sometimes the muscular sense is affected. The reflexes are usually normal; in some cases the knee-jerks are exaggerated, in a few cases they are diminished or even abolished.

There is no actual paralysis, but occasionally atrophy of the small muscles of the hand has been observed, and in rare cases general muscular atrophy has supervened. Fibrillary muscular tremors also occur. In the intervals between the attacks a remarkable increase of the mechanical and electrical excitability of the motor nerves may be observed. Stroking the side of the face will often produce contractions of the facial muscles; and the characteristic local spasms of the hands and feet may easily be started by compression of the nerves of the limb. The electrical irritability of the nerves is greater to the galvanic than to the faradic current; and the ulnar is commonly more irritable than any other nerve.

The morbid anatomy of tetany is still unknown. The nervous system has been partially examined in about forty cases. In fifteen of these the brain and spinal cord presented a normal appearance. Of the remaining cases changes in the brain are mentioned in six cases, and consisted in hyperæmia of the membranes, effusion into the subarachnoid space, and a softened or œdematous condition of the brain substance. The most frequent and constant changes have been observed in the cord; they are mentioned in seventeen cases, and comprise hyperæmia of the cord and its membranes, softening of the substance, especially in the cervical and lumbar enlargements, together with capillary hæmorrhages and accumulation of lymphoid cells around blood vessels. Langenhans, in one case, found thickening of the walls of the small arteries and veins, especially in those of the cervical enlargement, but the patient had reached an age—namely 48—when such changes are not uncommon. Weiss found marked changes in the ganglion cells of the anterior horns in three cases in which tetany had followed excision of the thyroid gland. In one case, three months after the operation, some of the cells in the cervical enlargement were swollen and contained vacuoles; others were atrophied, their nuclei were small and flattened, and had lost their processes; further, the axis cylinders of the fibres composing the anterior roots presented spindle-shaped swellings. Similar changes were found in the other two cases, but the ganglion cells were not wasted.

The peripheral nerves were microscopically examined in two cases (Berger and Weigert) and found healthy.

Pathology.—It is obvious that the results of post-mortem examination do not throw much light on the pathology of tetany. The morbid anatomy demands re-investigation, and it is especially important that a careful histological examination should be made of all parts of the nervous system in cases where tetany has existed for many years. At present, evidence as to the probable locality and nature of the lesion can only be obtained from a consideration of the ætiology and clinical aspects of the disease.

I.—As to localisation. We have seen that muscular spasm is chiefly met with in affections of the upper segment of the motor tract. In favour of the cerebral origin of tetany the following facts may be mentioned:—(1) That interosseous spasm occurs in some forms of brain disease, as, for example, in the spastic hemiplegia of infancy. (2) That tetany in infants is frequently associated with laryngismus stridulus, and is occasionally followed by general convulsions. (3) That headache, vertigo, and even psychical disorders have been observed; while bulbar disturbance has sometimes been suggested by the presence of polyuria and glycosuria.

Against the cerebral origin of tetany, according to Abercrombie, is the persistence of the tonic spasms during sleep and chloroform anæsthesia. If, too, the spasms depended on cortical disturbance we should have to admit the limitation of this disturbance, in many cases at least, to certain portions of the hand and foot centres. It may also be incidentally noticed that the position of the hand in tetany differs from that seen in the eclamptic attacks of infancy. In both there is rigid flexion, but in eclampsia all the joints of the fingers are flexed, and the thumbs, instead of being pressed against the forefingers, are flexed and drawn inwards under cover of the firmly bent fingers. Moreover, the convulsions which sometimes occur during an attack of tetany do not appear to have any influence over it. The features of tetany persist through the convulsive attack, and although both are probably started by the same irritant, their independence suggests that different parts of the nervous system are being excited.

As to the upper segment of the motor tract in the cord, it may be observed that a spastic weakness of arms and legs sometimes occurs in consequence of degeneration of the pyramidal tracts, and when unattended by muscular atrophy or sensory disturbance is regarded by some authorities to point to a primary lateral sclerosis. But in these cases a careful examination will usually reveal impairment of the superficial abdominal reflexes or other indications that the grey matter of the cord is also implicated. Moreover, ankle clonus is present, and the knee-jerks and tendon reactions in the arms are exaggerated, phenomena which if they occur in tetany must be very rare.

Coming now to the lower segment of the motor tract, we have already pointed out our ignorance with regard to the occurrence of spasmodic conditions in connection with lesions of the anterior horns. Even physiological experiments do not give us much assistance. The spasms produced by strychnine are said to be due to the action of the poison on the spinal grey matter, but we are not aware that there is any direct proof of this. All that can be said is that strychnine increases the excitability of some portion of the reflex arc.

The occasional occurrence of muscular atrophy in cases of tetany may be due to a lesion of the ganglion cells of the anterior horns, but it may also be accounted for by disease of the peripheral nerves. Other points too bear testimony to the possibility of the peripheral origin of tetany. They are:—(1) The distribution of the disorder—in ordinary cases the hands and feet are the only parts of the body affected—which presents a resemblance to that of alcoholic neuritis. The extremities are flexed by spasm in tetany, they are flexed in consequence of weakness in alcoholic paralysis. (2) Evidence as to disturbance of sensory fibres, namely, paræsthesia and rarely anæsthesia. Also the presence of Hoffmann's phenomenon, namely, exaggerated excitability of the sensory nerves to mechanical and electrical stimulation. (3) The increased mechanical and electrical excitability of the motor nerves, as well as the occasional presence of fibrillary muscular contractions, of muscular paresis, and atrophy.

II.—What is the nature of the irritant? Without entering into a full discussion of the ætiology of tetany, two pathological facts of great significance may be briefly noticed. (1) That tetany is frequently associated with some disturbance of the alimentary canal. (2) That it often follows excision of the thyroid gland.

When present in infancy tetany is nearly always found in cases of rickets, but as a rule only in those cases where there are offensive stools, or other evidence of bad digestion. In adults tetany has been observed in cases of dilatation of the stomach. Bouveret and Devic have recently written a valuable paper on this subject, in which they have collected 23 cases. From a number of exhaustive experiments and analyses, they conclude: (*a*) that the tetany which occurs in patients affected with gastric dilatation is almost exclusively limited to cases where there is permanent hypersecretion of the gastric juice; (*b*) tetany is not produced reflexly by irritation of the stomach, but by the direct action of a chemical poison on the nervous system; (*c*) that the toxic substance is produced by the action of free hydrochloric acid on syntonin, and by the action of alcohol on that; (*d*) that the introduction into the blood of any animal of a solution of an alcoholic extract of the digestive fluids produces violent tetanic convulsions; but the latter do not occur when the injection is made subcutaneously.

The occurrence of tetany after extirpation of the thyroid gland is difficult to explain. It has been proved that tetany does not follow other operations in the neck, and that it follows total rather than partial removal of the thyroid. The association of tetany with myxœdema after the operation suggests that possibly the tonic spasms are excited by the poisonous effects of mucin, and it is significant that the injection of mucin into cats has sometimes been followed by tetany (Wagner and Hammerschlag). The probability that tetany, in a large number of cases is due to the irritating effects of some poison in the blood is not only indicated by the facts of its relation to alimentary disturbance and to excision of the thyroid gland, but also by its occurrence after some of the acute specific fevers, after lead poisoning, and by the phenomena of ergot poisoning, which are almost identical with those of tetany.

It appears, then, reasonable to assume that the condition called tetany is due to irritation of some portion of the nervous system by the presence of a poison in the blood or lymph; and that the facts connected with the ordinary features of the disease are perhaps best explained on the supposition that motor nerve fibres and not nerve cells are selected by the poison. At the same time the action of the poison is not limited to the peripheral nerves, for symptoms indicating central disturbance are sometimes combined with those which, as just suggested, appear to be related to a morbid condition of the peripheral nerves.

LOTHAR V. FRANKL-HOCHWART. "Die Tetanie."—Berlin, 1891. This thesis contains a full bibliographical index of publications on Tetany between the years 1830 and 1890. The following are some of the chief references to the subject since Hochwart's thesis:—

CHVOSTEK (F.) "Ueber das Verhalten der sensiblen Nerven, des Hörnerven und des Hautleitungswiderstandes bei Tetanie."—*Zeitsch. f. klin. Med.*, 1891, XIX., 489.

GANGHOFNER (F.) "Ueber Tetanie im Kindesalter."—*Zeitschr. f. Heilk.*, 1891, XII., 447.

SCHLESINGER (H.) "Ueber einige Symptome der Tetanie."—*Zeitsch. f. klin. Med.* 1891, XIX., 468.

RÉMOND (A.) "La tétanie."—*Gaz. d. Hôp.*, 1891, LXIV., 1225.

BOUVERET et DEVIC. "Sur la pathogénie de la tétanie d'origine gastrique."—*Compt. rend. Soc. de Biol.*, 1891, 9 S., III., 823; also *Revue de Méd.*, 1892.

RETTIG (A.) "Ein Fall von einseitiger Tetanie; Heilung."—*Ber. d. k. k. Krankenanst.* Rudolph-Stiftung in Wien, 1890, 297.

SCHLESINGER (H.) "Versuch einer Theorie der Tetanie."—*Neurol. Centralbl.*, 1892, XI., 66.

Professional hyperkineses.—It is not my intention to enter into a discussion of the pathology of the various occupation neuroses, because even where there is evidence of neuritis it is of a localised form, and this is a variety which scarcely demands more than brief notice in a work devoted to *multiple* neuritis.

In all probability this class comprises two groups of cases—the one group owning a peripheral, the other a central origin.

The former is constituted by cases in which certain muscles that have been subjected to overstrain become weak and wasted, and give altered reactions to electricity; the atrophic paralysis may or may not be associated with sensory disturbance. The latter comprises cases in which functional disturbances are exhibited, not in the performance of all the actions of the affected part, as in the first group, but only in the performance of special work.

As examples of the first group may be mentioned a case of sawyers' cramp, described by Dr. Poore, and a case of drummers' paralysis, recorded by Dr. Bruns. In the former case, the two muscles most strained in the act of sawing, namely, the supra-spinatus and the clavicular portion of the pectoralis major, were wasted, and the nerves supplying them were distinctly tender. In the latter case, the flexor longus pollicis and, to a less degree, the adductor pollicis of the left hand were paralysed and atrophied. The flexor longus pollicus could not be excited to contraction by either the galvanic or the faradic current, and, as Bruns points out, this muscle of the left hand is kept by the act of drumming in a state of constant contraction.

Examples of the second group are furnished by disorders of the special movements of writing. Now in many of the cases included under the term "writer's cramp," the most careful examination may fail to detect muscular spasm or weakness of the affected hand for any action other than that of writing. And when a purely acquired and complex movement such as that of writing becomes disordered while other movements of the hand and digits remain normal, it appears to me impossible to resist the conviction that nerve cells and not nerve fibres are the parts where the primary derangement is located. (See Gowers, Vol. I., pp. 666—670.)

POORE (G. V.) "On a case of sawyer's cramp."—*Brain*, 1883, VI., 233.

BRUNS (L.) "Isolirte Lahmung des linken Flexor pollicis longus durch Ueberanstringung: Trommerlahmung."—*Neurol. Centralbl.*, 1890, 359.

SOME POINTS IN THE GENERAL PATHOLOGY OF PERIPHERAL NEURITIS.

The work of the last two years, much of which has been reviewed in the preceding pages, has demonstrated in the clearest possible way the existence of a group of cases characterised by the presence of certain well-defined symptoms, and by the limitation of pathological changes to the peripheral parts of the spinal nerves, the spinal cord, roots, and proximal portions of the nerves being often quite healthy. In applying the term peripheral neuritis to this group it must be clearly understood that the word neuritis is not restricted to inflammation of a nerve, but that it includes degeneration, atrophy, or any other kind of disease affecting a nerve (see Buzzard, *Trans. of Path. Soc.*, 1889). It is in this wide sense that the term is used in the present work, namely, that the peripheral portions of the nerves are the chief parts of the nervous system to be affected by morbid action; they are in an abnormal state, not necessarily inflammation, while other parts of the nervous system are less constantly or specially affected.

The abnormal condition may be demonstrable on post-mortem examination, or, it is conceivable, may be so temporary or slight as to leave no changes that can be detected by the highest powers of the microscope. The inclusion, therefore, of many conditions under the heading peripheral neuritis can only be justified after a consideration of their clinical analogies with other conditions where the peripheral nerves show definite organic change, and I imagine that Dr. Ross based his classification on these lines, and included, for example, Landry's disease, tetany and Raynaud's disease, because the grouping of their symptoms more closely resembled that of peripheral than of central disease.

Perhaps the most characteristic clinical feature of multiple neuritis is symmetrical localisation of motor and sensory symptoms to the peripheral parts of the limbs. The chief motor symptom is weakness of the extensor muscles of the hands, and of the dorsal flexors of the feet; while the sensory symptoms comprise diminution and perversion of cutaneous sensibility, together with tenderness of the nerve trunks and muscles. Such being the case, it appears not unreasonable to suppose that other symptoms than the classical ones, if they present a similar distribution, may be dependent on an abnormal state of the peripheral nerves. In other words, if in place of paralysis, muscular spasms or vascular disturbances form the dominant features, but have the same symmetrical localisation to the extremities, is it not probable that they too may be due to peripheral neuritis? These questions have been partially discussed in connection with tetany and Raynaud's disease. But while admitting the importance in diagnosis of symmetrical localisation to peripheral parts, we must remember how widely the symptoms of multiple neuritis vary, both in character and distribution, and how difficult it is in some cases to decide whether the lesion is situated in the cord or in the nerves. Instead of a symmetrical we may have a random distribution of symptoms in peripheral neuritis; instead of evidence that many nerves are affected (as we should expect from the presence of a poison in the blood), the symptoms may be located to the territory of a plexus, or to that of a particular nerve. Buzzard and Brissaud have each drawn attention to localised forms of alcoholic neuritis; Leudet to a case in which only the muscular branches of one ulnar nerve were affected; whilst isolated neuritis has also been observed in connection with diabetes, influenza, and other diseases.

The difficulty in diagnosis is further illustrated by cases of widespread atrophic paralysis, such as those alluded to in connection with syphilis, influenza, and lead poisoning. A remarkable example of this difficulty is afforded by the history and post-mortem examination of a case, published in great detail by MM. Blocq and Marinesco. In this case the illness had extended over a period of twenty years. The attack was ushered in by fever and digestive derangements; there was generalised atrophic paralysis, which was succeeded by local recovery. Sensory symptoms were entirely absent, and the case was diagnosed as one of acute spinal paralysis. The autopsy revealed great degeneration of the muscles and of the intra-muscular nerve branches; other parts of the nerves were healthy, and only senile changes were found in the spinal cord.

This case proves that a generalised muscular atrophy may be dependent on neuritis, while it suggests that even where many peripheral nerves are implicated a toxic agent may fasten upon and have its action limited to motor fibres. Blocq and Marinesco point out that multiple neuritis was indicated in the case reported by the presence of psychical phenomena, diarrhœa, vomiting, œdema, and vaso-motor derangements.

What, then, is the explanation of the variations in the distribution and character of the symptoms met with in multiple neuritis? The commonest cause of the disease is admitted to be a chemical poison, which may be assumed to circulate freely in the blood and to be conveyed to all parts of the nervous system. Its frequent action on the brain is shown by the prevalence of psychical phenomena; the extent of its action on the spinal cord is not easy to define, but that it directly attacks the peripheral nerves can scarcely be doubted. The question naturally arises, are variations in the motor and sensory phenomena to be explained by differences in the selective action of the poison on nerve fibres or on the ganglionic cells which preside over their nutrition? In other words, what influence does the central nervous system exercise on the distribution and character of the symptoms presented by peripheral neuritis?

There are three possible ways in which the limitation of changes to the peripheral nerves may be explained:—(1) The poison selects and attacks those parts solely or predominantly; (2) it primarily attacks nerve cells, and, as a consequence, those portions of the nerve fibres, viz., the peripheral, which are furthest removed from their influence undergo degeneration; (3) the poison acts with equal intensity on nerve cells and nerve fibres; the former recover, but the latter, having been robbed for a time of vitality, have lost resisting power, and degeneration, already started, steadily progresses.

At first sight it appears reasonable to conclude that if the peripheral branches of nerves are alone diseased, the poison has singled them out for attack, and has had no affinity for nerve-trunks, cord, or brain. But we are faced by the difficulty that occasionally a trophic change, as muscular atrophy, is met with apart from demonstrable lesions in any part of the nervous system but where there are reasons for supposing that it depends on central disturbance. Two instances of this are particularly suggestive—arthritic muscular atrophy and the atrophy which rarely affects hemiplegic limbs. Now, as regards the former, the evidence is against neuritis, and is in favour of the hypothesis advanced by Charcot, namely, that the nutrition of the motor cells of the cord is deranged in a reflex manner, morbid impulses being conveyed to them from the joint nerves, and that their derangement or torpid condition determines the alterations in the muscles. That the process is a reflex one has indeed been demonstrated by Raymond, who found that a previous division of the posterior spinal roots prevented the wasting of the muscles.

The occurrence of muscular atrophy in hemiplegia, and sometimes (as in a case recorded by Babinski) when no spinal or neurotic changes can be discovered, suggests that the cerebral cortex may play a rôle in the dispensation of trophic lesions. This is also indicated by the occurrence of trophic lesions in limbs affected by hysterical paralysis, and further by such cases as those related by Dr. Bristowe under the heading of hysterical peripheral neuritis.

But while it is difficult to exclude central influences from an explanation of the phenomena of neuritis, it may be granted that restriction of pathological changes to peripheral nerves cannot be wholly explained by lowered vitality of central cells, but must be due, at least in part, to a selective and preponderating action of the poison on the nerves; and if it be admitted that in certain cases a poison exerts its greatest influence on the peripheral portions of the nervous system, it is logical to infer that cases exist in which these parts are alone attacked, the central portions of the nervous system presenting no affinity for the particular poison. The words of Graves, uttered nearly forty years before the days of peripheral neuritis, may be fitly quoted. In his introductory remarks to "Diseases of the Nervous System" he says:—"In considering the symptoms that accompany diseases of the nerves, pathologists have directed their attention almost exclusively to the nervous centres, and have looked on the brain, cerebellum, and spinal cord as the parts in which the causes of all nervous disorders reside, or in which they originate, forgetting that these causes may be also resident in the nervous cords themselves, or their extremities. In fact, gentlemen, pathologists have, with respect to the diseases of the nervous system, continued an error precisely similar to that which was so long prevalent with regard to diseases of the vascular system; for it is only lately that they have recognised the important truth that diseased vascular action may commence in the circumference." "And may not," he says in another passage, "the decay and withering of the nervous tree commence occasionally in its extreme branches? And may not a blighting influence affect the latter, while the main trunk remains sound and unharmed."

The view that nerves may alone be picked out by certain poisons receives further support from the frequent implication of sensory fibres in cases of peripheral neuritis. These fibres are found diseased when no changes can be discovered either in the cord or the posterior ganglia.

In conclusion, some of the main pathological features of peripheral neuritis may be summarised as follows:—(1) The chief cause is a chemical poison. (2) The poison affects all parts of the nervous system, but to a very unequal degree in different cases, partly because the nature of the poison varies, and partly because individual portions of the nervous system present varying susceptibilities in different persons. It may, indeed, be safely assumed that sometimes the peripheral nerves are solely implicated, while it is probable that particular fibres—motor, sensory, or vasomotor—may be picked out by special poisons. Nevertheless, in the majority of cases, evidence is usually forthcoming that the brain, or cord, or both, are attacked along with the nerves.

In all cases it must be remembered that although nerve fibres depend on nerve cells for their vitality, the condition of the cells is influenced by that of the fibres; not only may central changes, functional or organic, lead to peripheral lesions, but irritation of peripheral nerves may initiate lesions in nerve centres.

The pathology of peripheral neuritis needs to be placed on a broad basis. The nervous system must be regarded as a whole, and not as composed of segments artificially linked together; the functions of its various parts admit of no mathematical separation, and while the term peripheral neuritis is a convenient one to retain for a group of cases marked by the possession of certain definite clinical features, it must not be taken to imply that pathological changes are necessarily limited to the nerves. On every side, too, this group of cases merges into other groups, in some of which the central and peripheral portions of the nervous system are both diseased, whilst in other groups lesions appear to be strictly limited to the central parts.

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BLOCQ et MARINESCO. "Poliomyélitis et polynévrites."—*Nouvelle Iconographie de la Salpêtrière*, 1890, T. III., pp. 137, 195, 272.

JOFFROY et ACHARD. "Contribution à l'étude de l'atrophie musculaire chez les hémiplégiques."—*Arch. de Médéc. expérim. et d'Anat. pathol.*, 1891.

BRISTOWE. "Hysterical peripheral neuritis."—*Brit. Med. Journ.*, Nov. 19, 1892, p. 1098.

THE TREATMENT OF PERIPHERAL NEURITIS.

The first essential in dealing with any case of peripheral neuritis is to find out the cause and to remove it or stop its action as soon as possible. The cause, as we have seen, is nearly always some poison, and if the patient be withdrawn from its influence and placed under favourable conditions, complete recovery is the rule. There is abundant proof that muscles extremely atrophied as a result of neuritis may completely regain their normal bulk, strength, and electrical reactions. Hence it is clear that nerve fibres profoundly degenerated may be entirely reformed; new axis cylinders develop, and become covered with myelin, till ultimately a new set of nerve fibres convey healthy impulses to healthy muscles.

In cases due to alcohol the patient should be deprived at once from taking any form of alcoholic drink; even in old, broken-down topers the deprivation is rarely attended with danger if suitable nourishment be administered and careful attention be given to the condition of the digestive organs. To provide against duplicity on the part of the patient and to ensure the complete withdrawal of alcohol, it is often highly desirable to remove the patient from the care of his friends and to place him in the charge of trained and reliable nurses.

The treatment of neuritis will vary according to the stage of the disease, but in all except the slightest cases rest in bed is advisable. The patient is then protected from exposure to cold, the pernicious effects of movement are reduced to a minimum, while local treatment can be more readily and satisfactorily carried out.

1. In the acute stage the suffering of some patients is extreme, and it is of the utmost importance to relieve this as promptly and effectively as possible. The severest cases require a water-bed; this not only relieves pain better than an ordinary bed, but gives more support to a weak patient, and thus lessens the danger to life arising from a feeble dilated heart or paralysed respiratory muscles.

In feeding the patient or in attending to his evacuations the nurse should exercise the greatest care and gentleness, so that all unnecessary movements on the part of the patient may be avoided. For the relief of tender nerves and muscles there is nothing better than the application of warm fomentations. It is best to apply them intermittingly; a warm fomentation may be put on the painful part for half-an-hour, and the application repeated every four hours; a layer of hot cotton-wool taking its place in the intervals. Mills* recommends rapidly-alternating applications of very hot and very cold water; "a large sponge or soft towel is dipped first in very hot and another in very cold water, and one is made to rapidly follow the other up and down the limb." Occasional vapour baths often afford the patient great comfort, but they should not be used when the action of the heart is much impaired. As regards drugs, in the early stages of multiple neuritis, salicylate of soda and iodide of potassium, either alone or in combination, appear to be of service; Mills speaks highly, too, of oil of gaultheria. Neuralgic pains may also be relieved by the administration of antipyrin, phenacetin, or exalgin, but when the suffering is very great the hypodermic injection of morphia becomes necessary.

The drugs mentioned are of value in all forms of peripheral neuritis, but special treatment is called for in particular cases. Thus quinine must be given when paralysis is due to malarial poisoning; mercury and iodide of potassium in syphilitic cases, while perchloride of iron in large doses deserves a trial when the neuritis depends on anæmia, or on a septicæmic blood state. The weak dilated heart of alcoholism requires digitalis and strychnine, to which small doses of cocaine may often be added to lessen the craving for stimulants.

But of far greater importance than drugs is the regular and careful administration of nourishment, in the form of boiled milk, beef tea, beef extracts, soups and broths. Benger's food or peptonised gruel may be required, and when vomiting is a prominent symptom, nutrient enemata should be administered.

Complete rest in bed in a well-ventilated room, careful feeding, and exposure to sun light, are the essential points in the treatment of the early stages of multiple neuritis.

2. When the acute symptoms have subsided, recourse may be had to massage, electricity, and tonic treatment. Massage would be unendurable, and no doubt hurtful in the acute stage; but its application should not be delayed too long. It may be commenced as soon as extreme pain and tenderness have disappeared. At first massage should be employed in the gentlest manner, and only for short periods of time.

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* *International Medical Magazine*, February, 1892.

But as soon as the patient stands it well, it ought to be regularly and vigorously applied. The patient should also be encouraged to make voluntary movement against resistance, and other forms of Swedish movements can be employed with advantage. By these means the nutrition and strength of the muscles are improved, while any tendency to contractures is overcome. Moreover, the patient's capacity for assimilation of food is steadily increased.

The restoration of the degenerated nerves and muscles may also be aided by the daily application of galvanism. Large electrodes are desirable, and the current should be slowly interrupted. A daily warm bath, followed by vigorous friction to the skin, is of value, whilst tonics and cod-liver oil often prove of great service.

When should the patient be allowed to get up? Not until pain and tenderness have subsided and there is evidence that the process of repair has become established.

From first to last abundant fresh air and sunlight are of the greatest importance, and as soon as the patient is able to take outdoor exercise a change of air is often advisable; but in many cases it will be some time before local massage can be entirely dispensed with.

As to the efficacy of drugs in the elimination of poisons from the system, fresh investigations are needed. Some recent experiments by Dr. Dixon Mann, the results of which he has kindly permitted me to mention, appear to indicate that iodide of potassium has no appreciable influence on the elimination of lead. He made a systematic examination, extending over several months, of the urine and fæces taken from cases of lead poisoning, and found that no medicinal treatment had any effect on the rate of elimination of this poison. A certain proportion of lead forms definite combinations with organic matter, and may remain in the system for an indefinite time, but a small proportion undergoes progressive elimination independently of any treatment. Dr. Dixon Mann believes, however, that warm baths and general massage do contribute to a slight extent to increase the rate of normal elimination.

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JOHN HEYWOOD, Excelsior Printing and Bookbinding Works, Manchester